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O.M. BARWELL, RICHARD, 55, Wimpole Street, W. (C. 1872–5, V.P. 1877–9.) *Trans.* 21, *C.C.* 2.

O.M. BASTIAN, HENRY CHARLTON, M.D., F.R.S., 8A, Manchester Square, W. (C. 1876–8, V.P. 1891–3.) *Trans.* 7.

1896 BATTEN, FREDERICK E., M.D., 124, Harley Street, W. *Trans.* 2, *C.C.* 1.

1899 BATTEN, RAYNER DERRY, M.D., B.S., Campden Lodge, Campden Hill Road, W.

1888 BATTERHAM, JOHN WILLIAMS, M.B., B.S., Bank House, Grand Parade, St. Leonard's.

1886 BATTLE, WILLIAM HENRY, 49, Harley Street, W. (C. 1899– .) *Trans.* 7, *C.C.* 12.

1868 BÄUMLER, CHRISTIAN G. H., M.D., University of Erlangen. *Trans.* 4.

1896 BEATON, ROBERT MILNE, M.B., 7, Dartmouth Park Road, N.W.

1898 BEDDARD, ARTHUR PHILIP, M.B., 44, Seymour Street, Portman Square, W.

1880 **BEEVOR**, CHARLES EDWARD, M.D., 33, Harley Street, W. (C. 1890–1.) *Trans.* 2, *C.C.* 6.

1884 BENHAM, F. LUCAS, M.D., Campden House, nr. Exeter, Semaphore, South Australia. *Trans.* 4.

1883 BENHAM, ROBERT FITZROY, 11, Earl's Court Square, S.W.

1874 BENNETT, WILLIAM HENRY, 1, Chesterfield Street, W. (C. 1889, 1892–4, S. 1890–1, V.P. 1895–8.) *Trans.* 4, *C.C.* 3.

1896 BENTHAM, EDWARD COOPER, 10, Princes Street, Cavendish Square, W.

1889 BENTLEY, ARTHUR J. M., M.D., Mena House, Pyramids, Cairo, Egypt.

1896 BERKELEY, G. H. A. COMYNS, M.B., 53, Wimpole Street, W.

1882 BERRY, FREDERICK HAYCRAFT, M.B., Watford, Herts.

ELECTED

1885 BERRY, JAMES, B.S., 21, Wimpole Street, W. (C. 1898– .)

1889 BIDWELL, LEONARD ARTHUR, 59, Wimpole Street, W. *Trans.* 4, *C.C.* 1.

1882 BINDLEY, PHILIP HENRY, M.B. (Travelling).

1889 BISSHOPP, FRANCIS ROBERT BRYANT, M.A., M.B., B.S., Belle Vue, Mount Pleasant, Tunbridge Wells.

1887 BLAND-SUTTON, JOHN, 48, Queen Anne Street, W. (C. 1894-7.) *Trans.* 12. *C.C.* 3.

1893 BOND, JAMES WILLIAM, M.D., 26, Harley Street, W. *C.C.* 2.

1898 BOSANQUET, WILLIAM CECIL, M.D., 13, York Street, Portman Square, W.

1888 BOSTOCK, ROBERT ASHTON, Cefn Mor, Penmaen, Glamorganshire.

1899 BOWLAN, MARCUS MARWOOD, M.B., B.S., The Infirmary, Raine Street, St. George-in-the-East, E.

1883 BOWLBY, ANTHONY A., 24, Manchester Square, W. (C. 1891–3.) *Trans.* 3, *C.C.* 3.

1883 Bowles, ROBERT LEAMON, M.D., 16, Upper Brook Street, W. (C. 1890–1.)

1896 BOX, CHARLES R., M.D., St. Thomas's Hospital, S.E. *Trans.* 1.

1889 BOYD, STANLEY, M.B., 134, Harley Street, W. (C. 1899–.) *Trans.* 1, *C.C.* 6.

1890 BRADFORD, JOHN ROSE, M.D., F.R.S., 8, Manchester Square, W. (C. 1900– .) *Trans.* 2.

1878 BRIDGES, ROBERT, M.B., M.A., The Manor House, Yattendon, Berkshire. *Trans.* 1.

1868 BRIGHT, GEORGE CHARLES, M.D., Cannes, Alpes Maritimes, France.

O.M. BROADBENT, Sir WILLIAM HENRY, Bart., M.D., F.R.S., 84, Brook Street, W. (C. 1871–3, V.P. 1881–3, P. 1887–8.) *Trans.* 17.

1887 BROCK, J. H. E., M.D., B.S., 115, Adelaide Road, N.W.

1894 BRODIE, C. GORDON, Fernhill, Wootton Bridge, Isle of Wight. *C.C.* 1.

1898 Brook, WILLIAM HENRY BREFFIT, M.D., B.S., 8, Eastgate, The Close, Lincoln.

1890 BROWN, WALTER HENRY, 19, Queen Street, Leeds. *Trans.* 2.

1876 BROWNE, GEORGE BUCKSTON, 80, Wimpole Street, W. (C. 1891–3.) *Trans.* 3.

1893 BROWNE, JAMES WILLIAM, M.B., 37, Holland Park Avenue, W. *Trans.* 1.

1887 BROWNE, OSWALD AUCHINLECK, M.A., M.D., 7, Upper Wimpole Street, W.

1883 BRUCE, JOHN MITCHELL, M.D., 23, Harley Street, W.

1893 BRYANT, JOHN HENRY, M.D., 4, St. Thomas's Street, S.E. *Trans.* 1.

O.M. BRYANT, THOMAS, M.Ch., 65, Grosvenor Street, W. (C. 1872, V.P. 1876–7, P. 1885–6.) *Trans.* 8.

1890 BUCKLAND, FRANCIS O., B.A., M.B., C.M., Brockenhurst, Hants. *Trans.* 1.

ELECTED

1891 BURGHARD, FREDERIC FRANÇOIS, M.D., M.S., 86, Harley Street, W.

1881 BURNET, ROBERT WILLIAM, M.D., 36, Grosvenor Street, W. *Trans.* 1.

1879 BURTON, WILLIAM EDWARD, 24, Wimpole Street, W.

1881 BUTLIN, HENRY TRENTHAM, 82, Harley Street, W. (C. 1887–9.) *Trans.* 7, *C.C.* 1.

1884 BUXTON, DUDLEY WILMOT, M.D., B.S., 82, Mortimer Street, W.

O.M. BUZZARD, THOMAS, M.D., 74, Grosvenor Street, W. (S. 1870–2, C. 1873–6, V.P. 1880–1, P. 1895–7.) *Trans.* 15, *C.C.* 3.

1891 CADDY, ARNOLD, 2/2, Harington Street, Calcutta.

1886 CAHILL, JOHN, M.D., 12, Seville Street, Lowndes Square, S.W. *C.C.* 1.

1893 CALEY, HENRY ALBERT, M.D., 24, Upper Berkeley Street, W. *C.C.* 1.

1890 CALVERT, JAMES, M.D., The Warden's House, St. Bartholomew's Hospital, E.C. *Trans.* 1, *C.C.* 2.

1893 CAMPBELL, HARRY, M.D., 23, Wimpole Street, W., and Coniston Cottage, Fox Lane, Upper Norwood, S.E. *C.C.* 3.

1891 CARLESS, ALBERT, M.S., M.B., 10, Welbeck Street, W.

1890 CARR, JOHN WALTER, M.D., 19, Cavendish Place, W. *Trans.* 1, *C.C.* 4.

1885 CATON, RICHARD, M.D., 86, Rodney Street, Liverpool. *Trans.* 3.

O.M. CAYLEY, WILLIAM, M.D., 27, Wimpole Street, W. (C. 1874–5, S. 1876–8, C. 1879–80, V.P. 1885–6.) *Trans.* 8, *C.C.* 3.

1890 CHAPMAN, CHARLES WILLIAM, M.D., 21, Weymouth Street, W. (C. 1900– .) *Trans.* 1, *C.C.* 2.

1884 CHAPMAN, PAUL M., M.D., 1, St. John's Street, Hereford. *C.C.* 1.

1893 CHARSLEY, ROBERT STEPHEN, "The Old Barn," Slough.

1885 CHEYNE, W. WATSON, M.B., C.M., F.R.S., 75, Harley Street, W. (C. 1894–6.) *Trans.* 2, *C.C.* 8.

1873 CHISHOLM, EDWIN, M.D., Abergeldie, Ashfield, near Sydney, New South Wales.

O.M. CHURCH, WILLIAM SELBY, M.D., 130, Harley Street, W. (C. 1874–6.)

1873 CHURTON, THOMAS, M.D., 35, Park Square, Leeds. (C. 1889–91.) *Trans.* 6.

O.M. CLAPTON, EDWARD, M.D., Towercroft, 41, Eltham Road, Lee, S.E. (C. 1872–4.) *Trans.* 1.

1874 CLARK, ANDREW, 71, Harley Street, W. *Trans.* 1.

1887 CLARK, FRANCIS WILLIAM, Hong-Kong.

1898 CLARKE, JAMES JACKSON, M.B., 9, Old Cavendish Street, Cavendish Square, W. *C.C.* 4.

1885 Clarke, J. MICHELL, M.B., 28, Pembroke Road, Clifton, Bristol.

1885 CLARKE, WILLIAM BRUCE, M.B., 51, Harley Street, W. (C. 1894–7.) *Trans.* 2, *C.C.* 1.

1877 Clay, ROBERT HOGARTH, M.D., 4, Windsor Villas, Plymouth.

1887 CLEMOW, ARTHUR HENRY WEISS, M.D., C.M., 101, Earl's Court Road, W. *Trans.* 1, *C.C.* 1.

ELECTED

1877 **CLUTTON**, HENRY HUGH, M.A., 2, Portland Place, W. (C. 1885–7.) *Trans.* 12, *C.C.* 3.

1898 COBB, ERNEST HERBERT, 5, Cornwall Terrace, Regent's Park, N.W.

1893 COLE, ROBERT HENRY, M.D., Moorcroft, Hillingdon, Middlesex.

1882 COLLIER, HERBERT, M.D., The Grange, Gorleston, Great Yarmouth, Norfolk.

1896 COLMAN, WALTER STACY, M.D., 22, Wimpole Street, W. *Trans.* 2, *C.C.* 3.

1882 COLQUHOUN, DANIEL, M.D., Dunedin, New Zealand.

1868 COOPER, FRANK W. (address uncommunicated).

1899 COOPER, HARRY, M.D., Fownhope, Surbiton Hill, Surrey.

1880 COTTLE, WYNDHAM, M.D., 39, Hertford Street, W.

O.M. COUPER, JOHN, 80, Grosvenor Street, W. (C. 1874.)

1875 COUPLAND, SIDNEY, M.D., 16, Queen Anne Street, W. (S. 1882–4, C. 1885–7, V.-P. 1898– .) *Trans.* 5, *C.C.* 3.

1886 COUSINS, JOHN WARD, M.D., Riversdale, Kent Road, Southsea. *Trans.* 1.

1882 COXWELL, C. F., M.B., Brighton, Melbourne, Victoria. *Trans.* 2.

1897 CRAWFURD, RAYMOND H. P., M.D., 71, Harley Street, Cavendish Square, W. *C.C.* 2.

1879 CRIPPS, WILLIAM HARRISON, 2, Stratford Place, W. (C. 1886–8.) *Trans.* 3.

1872 CRITCHETT, G. ANDERSON, 21, Harley Street, W.

1877 CROCKER, HENRY RADCLIFFE, M.D., 121, Harley Street, W. (C. 1884–5.) *Trans.* 16.

1896 CROOKE-LAWLESS, W. R., Surg.-Maj. A.M.S., M.D., Guards' Club, S.W.

1896 CROSSE, WILLIAM HENRY, M.D., 45, Dover Street, W.

O.M. CROFT, JOHN, 6, Mansfield Street, W. (C. 1870–2, V.P. 1882–4.) *Trans.* 12.

1890 CROWLE, THOMAS HENRY RICKARD, 35, St. James's Place, S.W.

1898 CROUCH, HERBERT CHALLICE, 55A, Welbeck Street, Cavendish Square, W.

1893 CURTIS, HENRY JONES, M.D., B.S., 30, Harley Street, W.C. *C.C.* 1.

1872 DALBY, Sir WILLIAM BARTLETT, M.B., 18, Savile Row, W. (C. 1879–81.) *Trans.* 4.

1896 DALTON, NORMAN, M.D., 4, Mansfield Street, Portland Place, W. *Trans.* 1, *C.C.* 5.

1898 DARLEY-HARTLEY, WILLIAM, Warrenville, Cape Town, South Africa.

1893 DAUBER, JOHN HENRY, M.B., B.Ch., 29, Charles Street, Berkeley Square, W.

1886 DAVIES, ARTHUR T., M.D., 23, Finsbury Square, E.C. (C. 1897–1900.) *C.C.* 13.

1879 **Davy**, HENRY, M.D., 29, Southernhay, Exeter.

1889 DEAN, HENRY PERCY, M.B., 69, Harley Street, W.

1897 DEANE, JOHN HENRY, 50, Wallwood Road, Leytonstone, E.

1899 DE CHAZAL, EDMOND LUCIEN, M.D., Port Louis, Mauritius.

1879 **Dennis**, FREDERIC S., M.D., 542, Madison Avenue, New York, U.S.A.

ELECTED

1875 DENT, CLINTON T., 61, Brook Street, W. (C. 1884–6.) *Trans.* 5. *C.C.* 1.

O.M. DICKINSON, WILLIAM HOWSHIP, M.D., 9, Chesterfield Street, W. (C. 1874–5, V.P. 1888–9.) *Trans.* 2.

1891 DICKINSON, WILLIAM LEE, M.D., 9, Chesterfield Street, W. *Trans.* 7.

1894 **Dickson**, THOMAS HUGH, M.B., B.C., H.M. Customs, Lower Thames Street, E.C.

1871 DIVER, EBENEZER, M.D., "Garstone," St. Margaret's-on-Thames, Twickenham. (C. 1890–2.) *Trans.* 1.

1868 DRAGE, CHARLES, M.D., Hatfield, Herts.

1896 DREW, DOUGLAS, M.D., B.S., 39, Devonshire Street, Portland Place, W.

1879 DREWITT, F. G. DAWTREY, M.D., 14, Palace Gardens Terrace, Kensington, W. (C. 1886–8.) *Trans.* 2.

O.M. DUCKWORTH, Sir DYCE, M.D., LL.D. 11, Grafton Street, Bond Street, W. (C. 1875–7, V.P. 1887–8, P. 1891–2.) *Trans.* 19, *C.C.* 6.

1884 DUKE, EDGAR, 11, Wilbury Road, Hove.

1869 DUKE, OLLIVER THOMAS, M.B., Surgeon, Bengal Army, India.

1899 DUNCAN, ANDREW, M.D., 24, Chester Street, Grosvenor Place, S.W.

1889 DUNCAN, JOHN, M.D., St. Petersburg.

1889 DUNN, LOUIS ALBERT, M.S., The College, Guy's Hospital, S.E. (C. 1899– .) *Trans.* 2, *C.C.* 2.

1884 EDMUNDS, WALTER, M.C., 2, Devonshire Place, Portland Place, W. *Trans.* 2.

1899 EÏD, ALFRED, M.D., Cairo, Egypt.

1882 EMOND, EMILE, M.D., 113, Boulevard Beaumarchais, Paris.

1868 EVANS, JULIAN, M.B., 123, Finborough Road, Redcliffe Square, S.W.

1893 EVANS, WILLMOTT H., 13, Taviton Street, Gordon Square, W.C. *C.C.* 1.

1888 **EVE**, FREDERIC S., 125, Harley Street, Cavendish Square, W. (C. 1899– .) *Trans.* 4, *C.C.* 10.

1877 EWART, WILLIAM, M.D., 33, Curzon Street, W. (C. 1884–6.) *Trans.* 2, *C.C.* 2.

1899 FAGGE, CHARLES HERBERT, M.S., 22, St. Thomas's Street, London Bridge, S.E.

1868 **Fairbank**, FREDERICK ROYSTON, M.D., Westcott, Dorking, Surrey. *Trans.* 2.

1889 FARDON, EDWARD ASHBY, Middlesex Hospital, W.

1885 FENN, EDWARD LIVEING, M.D., Grey Friars, Colchester.

1887 FENWICK, E. HURRY, 14, Savile Row, W. *Trans.* 2, *C.C.* 2.

1872 FENWICK, J. C. J., M.D., Long Framlington, Morpeth.

1893 FENWICK, WILLIAM SOLTAU, M.D., 8, Devonshire Street, W. *Trans.* 2.

1878 FIELD, GEORGE P., 34, Wimpole Street, W.

1876 FINLAY, DAVID WHITE, M.D., 2, Queen's Terrace, Aberdeen. (C. 1885–7, S. 1891.) *Trans.* 7.

1894 FLETCHER, HERBERT MORLEY, M.D., 98, Harley Street, W. *Trans.* 1.

1878 **FONMARTIN**, HENRY DE, M.D., 26, Newberry Terrace, Lower Bullar Street, Nichols Town, Southampton.

1889 FORBES, DANIEL MACKAY, Shoreditch Infirmary, 204, Hoxton Street, N.

ELECTED

1890 FORMAN, E. BAXTER, M.D., 11, Bramham Gardens, S. Kensington, S.W.
1890 FOSTER, MICHAEL G., M.B., M.A., Villa Camilla, San Remo, Italy.
1898 FOWLER, CHARLES OWEN, M.D., Cotford House, Thornton Heath, Surrey.
1881 FOWLER, JAMES KINGSTON, M.D., 35, Clarges Street, W. (C. 1887–9, 1899–, S. 1896–9.) *Trans.* 4, *C.C.* 6.
1886 FOX, R. HINGSTON, M.D., 23, Finsbury Square, E.C. *C.C.* 1.
1878 FOX, THOMAS COLCOTT, M.B., 14, Harley Street, W. (C. 1888–90.) *Trans.* 7, *C.C.* 2.
1893 FOXWELL, ARTHUR, M.D., 22, Newhall Street, Birmingham.
1898 FRANK, PHILIP, M.D., 3, Elvaston Place, Queen's Gate, S.W.
1897 FRIPP, ALFRED DOWNING, M.S., 19, Portland Place, W. *Trans.* 3.
1899 FÜRTH, KARL, M.D., 94, Harley Street, W.
1890 FULLER, HENRY ROXBURGH, M.D., 45, Curzon Street, W.
1897 FURNIVALL, PERCY, 28, Weymouth Street, Portland Place, W.
1891 FYFFE, W. KINGTON, M.B., B.C., 19, Duke Street, Manchester Square, W. (Travelling, New Zealand.) *Trans.* 1.
1888 GAGE-BROWN, CHARLES HERBERT, M.D., 85, Cadogan Place. S.W.
1895 GALLOWAY, JAMES, M.D., 54, Harley Street, W.
1887 GARROD, ARCHIBALD EDWARD, M.A., M.D., 9, Chandos Street, Cavendish Square, W. (C. 1898– .) *Trans.* 3, *C.C.* 3.
1879 GARSTANG, THOMAS WALTER HARROPP, Headingley House, Knutsford, Cheshire.
1885 GIBBONS, ROBERT ALEXANDER, M.D., 29, Cadogan Place, S.W. *Trans.* 1.
1893 GIBBS, CHARLES, Charing Cross Hospital, W.C.
1875 GILBART-SMITH, T., M.A., M.D., 68, Harley Street, W. (C. 1883–5.)
1868 GLOVER, JAMES GREY, M.D., 25, Highbury Place, N. (C. 1878–80. V.P. 1892–4.) *Trans.* 2.
1893 GLOVER, LEWIS G., M.B., B.C., 1, College Terrace, Fitzjohn's Avenue, N.W.
1875 GODLEE, RICKMAN JOHN, M.S., 19, Wimpole Street, W. (C. 1882–3, 1887–9, S. 1884–6, V.P. 1895–8.) *Trans.* 13, *C.C.* 8.
1882 GOLDIE, ROBERT WILLIAM, Hirst House, Hirst, Morpeth, Northumberland.
1878 GOLDING-BIRD, C. H., M.B., 12, Queen Anne Street, W. (C. 1887–9.) *Trans.* 17, *C.C.* 1.
1894 GOODALL, EDWARD WILBERFORCE, M.D., Eastern Hospital, Homerton, N.E. *Trans.* 5.
1875 GOODHART, JAMES FREDERIC, M.D., 25, Portland Place, W. (C. 1880–2, V.P. 1897–1900.) *Trans.* 14, *C.C.* 2.
1891 GOODMAN, ROGER NEVILLE, M.B., 3, Grove Crescent, Kingston-on-Thames.
1869 GOODRIDGE, HENRY FREDERICK AUGUSTUS, M.D., 10, Brock Street, Bath.
1882 GOODSALL, D. H., 17, Devonshire Place, W.
1877 GOULD, A. PEARCE, M.S., 10, Queen Anne Street, W. (C. 1885–7 1895–8, S. 1892–5, V.-P. 1898– .) *Trans.* 14, *C.C.* 9.

ELECTED

1875 GOWERS, Sir WILLIAM RICHARD, M.D., F.R.S., 50, Queen Anne Street, W. (C. 1881–2.) *Trans.* 5.

1891 GRANT, J. DUNDAS, M.D., 8, Upper Wimpole Street, W.

1868 GREEN, T. HENRY, M.D., 74, Wimpole Street, W. (C. 1877–9.) *Trans.* 2, *C.C.* 1.

1875 Greenfield, WILLIAM SMITH, M.D., 7, Heriot Row, Edinburgh. (C. 1881.) *Trans.* 3.

1893 GRIFFITH, WALTER SPENCER ANDERSON, M.D., 96, Harley Street, W.

1895 Grube, KARL, M.D., Neuenahr, Germany. *Trans.* 1.

1895 GUTHRIE, LEONARD GEORGE, M.D., 15, Upper Berkeley Street, W. *C.C.* 5.

1887 HABERSHON, SAMUEL HERBERT, M.D., 88, Harley Street, W. *C.C.* 1.

1875 HALE, C. D. B., 3, Sussex Place, W. *Trans.* 1.

1878 HALL, F. DE HAVILLAND, M.D., 47, Wimpole Street, W. (C. 1885–7.) *Trans.* 8, *C.C.* 5.

1889 HALSTEAD, GEORGE EZRA, M.D., B.S., Albion Hill House, Ramsgate.

1888 HANDFIELD-JONES, MONTAGU, M.D., 35, Cavendish Square, W.

1886 Handford, HENRY, M.D., 14, Regent Street, Nottingham. (C. 1893–4.) *Trans.* 8, *C.C.* 1.

1886 HARDIE, JAMES, M.D., 15, St. John Street, Manchester.

1890 HARPER, JAMES, M.D., 25, Rosary Gardens, South Kensington, S.W.

1872 HARRIS, HENRY, M.D., Trengweath, Redruth, Cornwall.

1889 HARRIS, HERBERT ELWIN, M.B., 13, Lansdown Place, Victoria Square, Clifton, Bristol. *Trans.* 1.

1881 HARRISON, CHARLES EDWARD, M.B., Grenadier Guards Hospital, Rochester Row, S.W.

1892 HARRISON, DAMER, 53, Rodney Street, Liverpool. *Trans.* 1.

1869 HAWARD, J. WARRINGTON, 57, Green Street, Grosvenor Square, W. (C. 1876–8, 1884–6, S. 1881–3, V.P. 1888.) *Trans.* 14.

1899 HAWKES, CLAUDE SOMERVILLE, Frescati, Ann Street, Brisbane, Queensland, Australia.

1886 HAWKINS, FRANCIS HENRY, M.D., 73, London Street, Reading. *Trans.* 4, *C.C.* 3.

1889 HAWKINS, HERBERT PENNELL, M.D., B.Ch., 56, Portland Place, W. (C. 1900– .) *Trans.* 1.

1890 HAWKINS-AMBLER, GEORGE ARTHUR, 30, Rodney Street, Liverpool.

1900 HAYWARD, JOHN ARTHUR, M.D., 17, Lingfield Road, Wimbledon, S.W.

1879 HENDERSON, GEORGE COURTENAY, M.D., Kingston, Jamaica, West Indies.

1882 HERON, GEORGE ALLAN, M.D., 57, Harley Street, W.

1884 HERRINGHAM, WILMOT PARKER, M.D., 13, Upper Wimpole Street, W. (C. 1894–7.) *Trans.* 5, *C.C.* 3.

1888 Hetherington, GEORGE HAYNES, 10, Museum Street, Ipswich.

1897 HILLIER, ALFRED P., M.D., C.M., 30, Wimpole Street, W.

ELECTED

1874 HOLDERNESS, WILLIAM BROWN, 15, Park Street, Windsor.

1868 **Holman**, CONSTANTINE, M.D., 26, Gloucester Place, Portman Square, W. (C. 1894–7.)

O.M. HOLMES, TIMOTHY, 6, Sussex Place, Hyde Park, W. (C. 1867–9, V.P. 1873–5.) *Trans.* 16.

O.M. HOLTHOUSE, CARSTEN, 38, Townshend Terrace, Richmond, Surrey. (C. 1870–2.) *Trans.* 8.

1878 HOOD, DONALD WILLIAM CHARLES, M.D., 43, Green Street, Park Lane, W. (C. 1891–3.) *Trans.* 1.

1884 HORSLEY, VICTOR, M.B., F.R.S., 25, Cavendish Square, W. (C. 1894.) *Trans.* 2, *C.C.* 1.

1895 HOUGH, CHARLES HENRY, Full Street, Derby.

1880 **HOVELL**, T. MARK, 105, Harley Street, W. *Trans.* 1.

1893 HOWARD, R. J. BLISS, M.D., 31, Queen Anne Street, W.

1876 HOWSE, HENRY GREENWAY, M.S., 59, Brook Street, W. (C. 1881–3, V.P. 1890–2.) *Trans.* 3.

1898 HULKE, SYDNEY BACKHOUSE, 162, Holland Road, Kensington, W.

1897 HUMPHRYS, CHARLES B., Eagle House, Blandford, Dorset.

1897 HUNT, GEORGE BERTRAM, M.B., 47, Albemarle Crescent, Scarborough. *Trans.* 1, *C.C.* 1.

1892 HUNTER, WILLIAM, M.D., 103, Harley Street, W.

O.M. HUTCHINSON, JONATHAN, F.R.S., 15, Cavendish Square, W. (C. 1867–8, V.P. 1875–6.) *Trans.* 14, *C.C.* 1.

1896 HUTCHINSON, JONATHAN, jun., 15, Cavendish Square, W. *Trans.* 3, *C.C.* 3.

1893 IONIDES, THEODORE HENRY, M.B., B.S., 30, Palmeira Square, West Brighton.

1883 JACKSON, GEORGE HENRY, Ashburton, Carew Road, Eastbourne.

O.M. JACKSON, J. HUGHLINGS, M.D., F.R.S., 3, Manchester Square, W. (C. 1872–3, V.P. 1887.) *Trans.* 2, *C.C.* 1.

1877 JACOBSON, WALTER HAMILTON ACLAND, M.B., M.Ch., 66, Great Cumberland Place, W. (C. 1890–2.) *Trans.* 2, *C.C.* 1.

1888 JAMES, JAMES THOMAS, M.D., 30, Harley Street, W.

1888 JAMISON, ARTHUR, M.D., C.M., 18, Lowndes Street, S.W.

1889 JOHNSON, RAYMOND, M.B., B.S., 11, Wimpole Street, W. (C. 1900– .) *Trans.* 4, *C.C.* 2.

1893 JOHNSTON, G. F., M.D., 3, Montagu Place, Montagu Square, W.

1878 JOHNSTON, WILLIAM, M.D., M.C., 16, Lonsdale Terrace, Upper Kent Street, Leicester.

1898 **Jones**, ROBERT, 11, Nelson Street, Liverpool. *Trans.* 2.

1872 JONES, THOMAS RIDGE, M.D., 4, Chesham Place, S.W. (C. 1892–3.)

1876 JORDAN, FURNEAUX, Harborne, Birmingham. *Trans.* 1.

1886 JULER, HENRY EDWARD, 23, Cavendish Square, W.

1878 KEETLEY, CHARLES ROBERT BELL, 56, Grosvenor Street, W. (C. 1895–8.) *Trans.* 2.

ELECTED

1898 KELLOCK, THOMAS HERBERT, 8, Queen Anne Street, Cavendish Square, W. *Trans.* 2, *C.C.* 2.

O.M. KELLY, CHARLES, M.D., Ellesmere, Gratwicke Road, Worthing, Sussex.

1882 KESTEVEN, WILLIAM HENRY, Hillwood, Waverley Grove, Hendon, N.W. *Trans.* 2.

1897 KEYSER, CHARLES RALPH, 29, Hamilton Terrace, N.W.

1883 KIDD, PERCY, M.D. (*Hon. Secretary*), 60, Brook Street, W. (C. 1891–2. S. 1899– .) *Trans.* 4, *C.C.* 3.

1887 **Knaggs**, R. LAWFORD, B.C., 39, Park Square, Leeds. *Trans.* 2.

1878 LACEY, THOMAS WARNER, 196, Burrage Road, Plumstead, S.E.

1897 LAMPLOUGH, CHARLES, Chatteris, Cambridgeshire.

1890 LANCASTER, ERNEST LE CRONIER, M.B., B.Ch., Winchester House. Swansea, S. Wales. *Trans.* 2.

1895 LANE, JAMES ERNEST, 46, Queen Anne Street, W. *Trans.* 1.

1883 LANE, WILLIAM ARBUTHNOT, M.B., M.S., 21, Cavendish Square, W. (C. 1893–6.) *Trans.* 29, *C.C.* 10.

O.M. LANGTON, JOHN, 62, Harley Street, W. (C. 1878–80, V.P. 1892–4, P. 1897–9.) *Trans.* 5.

1886 LANKESTER, HERBERT, M.D., Ingleborough, Woking, Surrey.

1885 LARDER, HERBERT, Whitechapel Infirmary, Vallance Road, N.E. *C.C.* 1.

1893 LAWSON, ARNOLD, M.D., 12, Harley Street, W.

O.M. LAWSON, GEORGE, 12, Harley Street, W. (S. 1871–3, C. 1874–6, V.P. 1881–3.) *Trans.* 16.

1896 **Leech**, PRIESTLEY, M.D., King Cross, Halifax.

1877 **Lediard**, HENRY AMBROSE, M.D., 35, Lowther Street, Carlisle. (C. 1889.) *Trans.* 5, *C.C.* 1.

1877 LEES, DAVID B., M.D., 22, Weymouth Street, W. (C. 1885.) *Trans.* 6.

1893 LENDON, EDWIN HARDING, M.B., 162, Holland Park Avenue, W.

1899 LESLIE, MURRAY, M.D., 53, Queen Anne Street, Cavendish Square, W.

1892 LEWIS, EDWARD JOHN, M.B., B.C., 74, Hamilton Terrace, N.W.

1895 LEWIS, ERNEST E., M.D., Springfield, Chelmsford.

1879 LICHTENBERG, GEORGE, M.D., 47, Finsbury Square, E.C.

1890 LITTLE, JOHN FLETCHER, M.B., 32, Harley Street, W. *C.C.* 5.

1868 LITTLE, LOUIS STROMEYER, Shanghai, China.

1891 LITTLEWOOD, HARRY, 40, Park Square, Leeds. *Trans.* 1.

1875 LIVEING, EDWARD, M.D., 52, Queen Anne Street, W.

1885 LOCKWOOD, CHARLES BARRETT, 19, Upper Berkeley Street, W. (C. 1896-9.) *Trans.* 6.

1876 LONGHURST, ARTHUR EDWIN TEMPLE, M.D., 4, Eaton Square, S.W. (C. 1889–91.) *Trans.* 1.

1897 LOW, HAROLD, M.B., 10, Evelyn Gardens, South Kensington, S.W.

1899 LOW, V. WARREN, M.D., B.S., 86, Oxford Terrace, Hyde Park, W.

1881 LUBBOCK, MONTAGU, M.D., 19, Grosvenor Street, W.

1876 LUCAS, R. CLEMENT, M.B., B.S., 50, Wimpole Street, W. (C. 1883–5.) *Trans.* 11. *C.C.* 1.

ELECTED

1894 LUFF, ARTHUR PEARSON, M.D., 31, Weymouth Street, W.

1879 LUNN, JOHN REUBEN, New Marylebone Infirmary, Rackham Street, Ladbroke Grove Road, W. (C. 1890–1.) *Trans.* 8, *C.C.* 13.

1893 LYS, HENRY GRABHAM, M.D., Southbrook, Suffolk Road, Bournemouth.

1889 MACBRIDE, P., M.D., 16, Chester Street, Edinburgh.

1871 MAC CORMAC, Sir WILLIAM, Bart., K.C.V.O., 13, Harley Street, W. (C. 1877–9, V.P. 1888–9.) *Trans.* 5.

1891 MACDONALD, GREVILLE, M.D., 85, Harley Street, W.

1881 MCHARDY, MALCOLM MACDONALD, 5, Savile Row, W. *Trans.* 1.

1882 MACKENZIE, FREDERIC MORELL, 29, Hans Place, S.W.

1891 MACKENZIE, HECTOR W. G., M.A., M.D., 59, Welbeck Street, W. *Trans.* 6, *C.C.* 1.

1879 MACKENZIE, STEPHEN, M.D., 18, Cavendish Square, W. (C. 1884, 1888–90, S. 1885–7. V.P. 1900– .) *Trans.* 9, *C.C.* 10.

1884 MACKERN, JOHN, M.B., St. German's Lodge, Shooter's Hill Road, Blackheath, S.E.

1879 MACLAGAN, THOMAS JOHN, M.D., 9, Cadogan Place, S.W. (C. 1889–91.) *Trans.* 3.

1885 MACLAREN, RODERICK, M.D., Portland Square, Carlisle. *Trans.* 1.

1896 MAC RAE, FARQUHAR, M.B., 27A, Lowndes Street, Belgrave Square, S.W.

1898 MACREADY, JONATHAN F. C. H., 42, Devonshire Street, Portland Place, W.

1879 MAGILL, JAMES, M.D., M.C., Coldstream Guards, Queen Anne's Mansions, S.W.

1885 MAGUIRE, ROBERT, M.D., 4, Seymour Street, W. *Trans.* 1.

1881 MAKINS, GEORGE HENRY, 47, Charles Street, Berkeley Square, W. (C. 1892–4, 1898– , S. 1895–8.) *Trans.* 4, *C.C.* 7.

1887 MALCOLM, JOHN D., M.B., C.M., 13, Portman Street, W. (C. 1900– .) *Trans.* 1, *C.C.* 1.

1890 MANSON, PATRICK, M.D., C.M., 21, Queen Anne Street, W. (C. 1895–7.)

1888 Marriott, HYDE, M.B., Dial House, Stockport.

1868 MARSH, F. HOWARD (*Treasurer*), 30, Bruton Street, W. (C. 1876–7, 1881–3, S. 1878–80, V.P. 1887–8, T. 1899– .) *Trans.* 16, *C.C.* 6.

1887 MARTIN, SIDNEY, M.D., B.S., F.R.S., 10, Mansfield Street, W. (C. 1896–9.)

1888 MASON, DAVID JAMES, M.D., C.M., Rosemont, Maidenhead.

1892 MASTERS, JOHN ALFRED, M.D., 31, Albert Gate, Hyde Park, S.W.

1884 MAUDSLEY, HENRY CARR, M.D., 22, Collins Street, Melbourne, Victoria.

1897 MAY, CHICHESTER GOULD, M.D., 59, Cadogan Place, S.W.

1868 May, EDWARD HOOPER, M.D., High Cross, Tottenham, Middlesex.

1888 MAY, WILLIAM PAGE, M.D., B.Sc., 49, Welbeck Street, W.; and Helouan, nr. Cairo, Egypt (October to April).

1888 MENZIES, J. HERBERT, 47, Earl's Court Square, S.W.

1893 MERCER, WILLIAM BRACEWELL, M.B., B.C., Hospital for Sick Children, Moor Edge, Newcastle-on-Tyne.

ELECTED

1878 MEREDITH, WILLIAM APPLETON, C.M., 21, Manchester Square, W. (C. 1887–9.) *Trans.* 2.

1894 MICHELS, ERNST, M.D., 48, Finsbury Square, E.C.

1873 MICKLE, WILLIAM JULIUS, M.D., Grove Hall Asylum, Bow, E. (C. 1897–1900.)

1890 MILEY, MILES, M.A., M.B., 21, Belsize Avenue, Hampstead, N.W.

1896 MILLS-ROBERTS, ROBERT HERBERT, Llanberis, North Wales. *Trans.* 1.

1882 MONEY, ANGEL, M.D., Hunter Street, Sydney, New South Wales. (C. 1888–90.) *Trans.* 3.

1874 MORGAN, JOHN HAMMOND, 68, Grosvenor Street, W. (C. 1883–5. V.P. 1898– .) *Trans.* 2, *C.C.* 8.

1888 MORISON, ALEXANDER, M.D., 14, Upper Berkeley Street, W. *Trans.* 2, *C.C.* 1.

1899 MORISON, RUTHERFORD, 14, Saville Row, Newcastle-on-Tyne. *Trans.* 1.

1877 MORRIS, HENRY, M.A., M.B., 8, Cavendish Square, W. (C. 1884–6.) *Trans.* 9.

1877 MORRIS, MALCOLM ALEX., 8, Harley Street, W. (C. 1890–2.) *Trans.* 1, *C.C.* 1.

1885 MOTT, FREDERICK WALKER, M.D., F.R.S., 25, Nottingham Place, W. (C. 1895–8.) *Trans.* 1, *C.C.* 4.

1899 MOUAT-BIGGS, CHARLES E. FORBES, 2, Fauconberg Villas, Bayshill, Cheltenham. *C.C.* 2.

1879 MOULLIN, CHARLES W. MANSELL, 69, Wimpole Street, W. (C. 1888–90.) *Trans.* 9, *C.C.* 4.

1899 MOYNIHAN, BERKELEY GEORGE ANDREW, M.S., 33, Park Square, Leeds.

1875 MURPHY, SHIRLEY F., 22, Endsleigh Street, Tavistock Square, W.C. (C. 1888–90.) *C.C.* 1.

1885 MURRAY, ALEXANDER DALTON, M.B., Colombo, Ceylon.

1893 MURRAY, GEORGE REDMAYNE, M.D., 11, Ellison Place, Newcastle-on-Tyne.

1883 MURRAY, HUBERT MONTAGUE, M.D., 25, Manchester Square, W. (C. 1893–6.) *Trans.* 1.

1894 MURRAY, JOHN, 110, Harley Street, W. *C.C.* 1.

1872 MYRTLE, ANDREW S., M.D., 8, Park Parade, Harrogate. (C. 1892.)

1892 NASH, WALTER GIFFORD, 31, St. Peter's, Bedford.

1875 NETTLESHIP, EDWARD, 5, Wimpole Street, W. (C. 1881–2.) *Trans.* 2.

1889 Newman, D., M.D., 18, Woodside Place, Glasgow. *Trans.* 4.

O.M. NORTON, ARTHUR TREHERN, Leyfields Wood, Ashampstead, Berks. (C. 1874–6.) *Trans.* 7.

O.M. NUNN, THOMAS WILLIAM, 8, Stratford Place, W. (C. 1873–4.) *Trans.* 9.

1880 O'CONNOR, BERNARD, M.D., 25, Hamilton Road, Ealing, W. *Trans.* 1.

1899 OGLE, CYRIL, M.B., 96, Gloucester Place, Portman Square, W. *Trans.* 1.

O.M. OGLE, JOHN WILLIAM, M.D., 96, Gloucester Place, Portman Square, W. (C. 1867–8, V.P. 1884–5.) *Trans.* 6.

1868 Ogle, WILLIAM, M.D., The Elms, Driffield Road, Derby.

ELECTED

1887 Oliver, THOMAS, M.D., 7, Ellison Place, Newcastle-upon-Tyne.

1887 OPENSHAW, THOMAS HORROCKS, M.B., 16, Wimpole Street, W. *C.C.* 1.

1868 OPPERT, FRANZ, M.D., 128, Leipzigerstrasse, Friedenau, Germany. *Trans.* 1.

1877 ORD, WILLIAM MILLER, M.D., 37, Upper Brook Street, W. (C. 1882–4, T. 1889–99.) *Trans.* 9, *C.C.* 1.

1889 ORD, W. WALLIS, M.D., B.Ch., The Hall, Salisbury. *Trans.* 1, *C.C.* 1.

1887 ORMEROD, JOSEPH ARDERNE, M.D., 25, Upper Wimpole Street, W. (C. 1899– .)

1884 ORMSBY, LAMBERT HEPENSTAL, M.D., 92, Merrion Square West, Dublin.

1883 ORTON, GEORGE HUNT, M.B., 1A, Campden Hill Road, Kensington, W.

1877 OWEN, ISAMBARD, M.D., 40, Curzon Street, W. (C. 1888–90.) *Trans.* 1.

1888 OXLEY, ALFRED RICE, M.D., Conisbro, St. Julian's Road, Leigham Court Road, Streatham Common, S.W.

1888 PAGE, FREDERICK, M.D., 1, Saville Place, Newcastle-on-Tyne.

1875 PAGE, HERBERT W., M.C., M.B., 146, Harley Street, W. (C. 1882–4, V.P. 1899– .) *Trans.* 7, *C.C.* 2.

1884 PAGET, STEPHEN, 70, Harley Street, W. (C. 1898– .) *Trans.* 6, *C.C.* 2.

1873 PARKER, ROBERT WILLIAM, 13, Welbeck Street, W. (C. 1882–4, 1890–2, S. 1887–9, V.P. 1893–6.) *Trans.* 15, *C.C.* 7.

1881 PARKER, RUSHTON, M.B., B.S., 59, Rodney Street, Liverpool. (C. 1896–9.) *Trans.* 1, *C.C.* 1.

1890 PARKIN, ALFRED, M.S., 24, Albion Street, Hull. *Trans.* 2.

1894 PARKINSON, JOHN PORTER, M.D., 57, Wimpole street, W. *C.C.* 2.

1898 PARTRIDGE, ARTHUR A. H., M.B., M.Ch., 117, Friern Barnet Road, New Southgate, N.

1881 PASTEUR, WILLIAM, M.D., 4, Chandos Street, W. (C. 1891–3.) *Trans.* 4, *C.C.* 4.

1893 PATERSON, DONALD ROSE, M.D., C.M., 18, Windsor Place, Cardiff.

1900 PATON, EDWARD PERCY, M.D., 84, Park Street, Grosvenor Square, W.

1892 PAUL, FRANK THOMAS, 38, Rodney Street, Liverpool. *Trans.* 1.

1893 PAUL, JOHN ERNEST, M.B., 26, Queensborough Terrace, Kensington Gardens, W. *Trans.* 1.

1883 PAUL, JOHN LISTON, M.D., 43, Queensborough Terrace, Kensington Gardens, W.

O.M. PAVY, FREDERICK WILLIAM, M.D., F.R.S., 35, Grosvenor Street, W. (C. 1869–71, V.P. 1882–4.) *Trans.* 3.

1886 PAYNE, JOSEPH FRANK, M.D., 78, Wimpole Street, W. (C. 1897–1900.) *Trans.* 1.

1879 PEEL, ROBERT, 120, Collins Street East, Melbourne, Victoria.

1886 Penny, WILLIAM JOHN, Coombe, West Crewkerne, Somerset.

1887 PENROSE, FRANCIS GEORGE, M.D., 84, Wimpole Street, W. (C. 1896–9.) *Trans.* 1, *C.C.* 1.

ELECTED

1882 PEPPER, AUGUSTUS JOSEPH, M.S., M.B., 13, Wimpole Street, W. *Trans.* 1.

1900 PERKINS, GEORGE STEELE, M.D., 85, Wimpole Street, W.

1898 PERKINS, J. J., M.D., 41, Wimpole Street, Cavendish Square, W. *Trans.* 1, *C.C.* 1.

1895 PHEAR, ARTHUR G., M.D., B.C., 47, Weymouth Street, Portland Place, W. *Trans.* 2.

1895 PHILLIPS, F. B. WILLMER, M.B., 7, Harper Place, Bedford.

1884 PHILLIPS, SIDNEY, M.D., 62, Upper Berkeley Street, W. (C. 1893–6.) *Trans.* 5, *C.C.* 4.

O.M. PICK, THOMAS PICKERING, 18, Portman Street, W. (S. 1874–7, C. 1878–80, V.P. 1885–6.) *Trans.* 4.

1885 PITT, GEORGE NEWTON, M.D., 15, Portland Place, W. (C. 1894–6.) *Trans.* 6, *C.C.* 4.

1883 PITTS, BERNARD, M.A., M.C., 109, Harley Street, W. (C. 1893.) *Trans.* 5.

1871 Playne, ALFRED, M.B., Maidenhead.

1884 POLAND, JOHN, 2, Mansfield Street, Cavendish Square, W.

1884 POLLARD, BILTON, B.S., 24, Harley Street, W. (C. 1895–8.) *Trans.* 2.

1868 POLLOCK, JAMES EDWARD, M.D., 52, Upper Brook Street, W. (C. 1878–80.)

1871 POORE, GEORGE VIVIAN, M.D., 32, Wimpole Street, W. (C. 1879–81.) *Trans.* 4.

1881 POWELL, H. A., M.A., 44, Sandgate Road, Folkestone.

O.M. POWELL, Sir R. DOUGLAS, Bart., M.D. (*President*), 62, Wimpole Street, W. (C. 1874–6, V.P. 1889–90, P. 1899- .) *Trans.* 4.

1868 PRENTIS, CHARLES, Surgeon-Major, Bengal Medical Service; India.

1884 PRINGLE, JOHN JAMES, M.B., 23, Lower Seymour Street, W. (C. 1890–2, 1897–1900.) *Trans.* 1, *C.C.* 3.

1884 PYE-SMITH, PHILIP HENRY, M.D., F.R.S., 48, Brook Street, W. (C. 1890–2.) *Trans.* 1.

1896 PYE-SMITH, RUTHERFORD JOHN, 350, Glossop Road, Sheffield.

1893 RAKE, ALFRED THEODORE, M.B., B.S., 8, Sheriff Road, West Hampstead, N.W.

1895 RAMSAY, HERBERT MURRAY, 35A, Hertford Street, Mayfair, W.

1889 RANKING, JOHN E., M.D., Hanover House, Tunbridge Wells.

1883 READ, THOMAS LAURENCE, 11, Petersham Terrace, Queen's Gate, S.W.

1889 Rennie, GEORGE EDWARD, M.D., College Street, Hyde Park, Sydney, N.S.W.

1868 RICE, MICHAEL W., M.D., Elmbank, Hargrave, Northamptonshire, (C. 1876–8.)

1898 RICHARDS, JOSEPH STEWART, M.D., B.S., The Infirmary, East Dulwich, S.E. *C.C.* 1.

O.M. RINGER, SYDNEY, M.D., F.R.S., 15, Cavendish Place, W. (C. 1871–2.) *Trans.* 1.

ELECTED

1896 RISDON, WILLIAM ELLIOT, M.D., 81, Chancery Lane, W.C.
1873 **Roberts**, DAVID LLOYD, M.D., 11, St. John Street, Manchester.
1888 ROBERTS, FRANK ERNEST, Tulse Dale Villa, Lower Norwood, S.E.
1883 ROBERTS, FREDERICK THOMAS, M.D., 102, Harley Street, W. (C. 1892–4.)
1900 ROBERTS, GRIFFITH LLOYD, M.B., C.M., Castle Square, Carnarvon, North Wales.
1890 ROBERTSON, ROBERT, M.D., Belgrave Road, Ventnor, Isle of Wight.
1885 ROBINSON, ARTHUR HENRY, M.D., Mile End Infirmary, Bancroft Road, N.E. *C.C.* 3.
1890 ROBINSON, GEORGE SOMERVILLE, Surgeon-Major, 13, Lupus Street, St. George's Square, S.W.
1892 ROBINSON, HENRY BETHAM, M.D., M.S., 1, Upper Wimpole Street, W. *Trans.* 6, *C.C.* 7.
1885 ROBSON, A. W. MAYO, 7, Park Square, Leeds. (C. 1893–6.) *Trans.* 13.
1897 ROGER-SMITH, HUGH, M.D., 1, College Terrace, Fitzjohn's Avenue, N.W. *Trans.* 1.
1889 ROLLESTON, HUMPHRY DAVY, M.A., M.D., 112, Harley Street, W. (C. 1899– .) *Trans.* 9. *C.C.* 2.
1888 ROPER, ARTHUR, "Colby," Lewisham Hill, Blackheath. *Trans.* 1.
1897 ROSS, FREDERICK WILLIAM FORBES, M.D., 15, Gower Street, W.C.
1877 **ROTH**, BERNARD, 38, Harley Street, Cavendish Square, and "Wayside," Preston Park Avenue, Brighton. *Trans.* 1, *C.C.* 4.
1890 ROUGHTON, EDMUND WILKINSON, B.S., 38, Queen Anne Street, W. *C.C.* 1.
1874 ROWLAND, EDWARD ROGER, Dordrecht, Wodehouse, S. Africa.
1898 RUSSELL, ALFRED ERNEST, M.D., B.S., Melton House, Manor Park, Lee, S.E.
1887 RUTHERFOORD, H. T., M.B., Salisbury House, Taunton.
1885 RYLE, REGINALD JOHN, M.D., Green View, Hadley Green, Barnet.
1882 SAINSBURY, HARRINGTON, M.D., 52, Wimpole Street, W. (C. 1898– .)
1899 SANDIFER, HENRY STEPHEN, M.D., B.S., 11, Porchester Gardens, W.
1893 SANSOM, ARTHUR ERNEST, M.D., 84, Harley Street, W. *Trans.* 1, *C.C.* 1.
1873 SAVAGE, GEORGE HENRY, M.D., 3, Henrietta Street, W. (C. 1882–3.)
1886 SAVILL, THOMAS DIXON, M.D., 60, Upper Berkeley Street, W. *Trans.* 1, *C.C.* 3.
1899 SCHORSTEIN, GUSTAVE, M.B., 11, Portland Place, W.
1886 SCOTT, ALFRED, 15, German Place, Brighton.
1894 SCOTT, BERNARD, "Hartington," Poole Road, Bournemouth.
1900 SCOTT, KENNETH, M.B., 38, Harley Street, W.
1892 SCOTT, RICHARD JAMES HERBERT, 28, Circus, Bath. *C.C.* 1.
1869 SEDGWICK, LEONARD WILLIAM, M.D., 48, Gloucester Terrace, Hyde Park, W. (C. 1879–81, V.P. 1897–1900.)
1897 SEGUNDO, CHARLES SEMPILL DE, M.B., B.S., 6, Brook Street, Hanover Square, W.
1892 SELWYN-HARVEY, JOHN STEPHENSON, M.D., 1, Astwood Road, S.W.

ELECTED

1878 SEMON, Sir FELIX, M.D., 39, Wimpole Street, W. (C. 1885–7.) *Trans.* 8, *C.C.* 3.

1884 SHARKEY, SEYMOUR J., M.D., 22, Harley Street, W. (C. 1895–8.)

1898 SHAW, HERBERT BATTY, M.D., University College Hospital, Gower Street, W.C. *C.C.* 2.

1889 SHAW, LAURISTON ELGIE, M.D., 64, Harley Street, W.

1875 SHERWOOD, ARTHUR PAUL, 8, Seaside Road, Eastbourne.

1886 SILCOCK, ARTHUR QUARRY, M.D., B.S., 52, Harley Street, W. (C. 1896–9.) *Trans.* 5. *C.C.* 2.

1879 SKERRITT, EDWARD MARKHAM, M.D., Edgecumbe House, Richmond Hill, Clifton, Bristol. (C. 1895–8.) *Trans.* 2.

1872 SLIGHT, GEORGE, M.D., 14, Old Burlington Street, W.

1882 SMITH, E. NOBLE, 24, Queen Anne Street, W. *Trans.* 1.

1896 SMITH, EBENEZER STANLEY, M.D., 68, Wimpole Street, Cavendish Square, W.

1888 SMITH, FREDERICK J., M.D., 138, Harley Street, W. *Trans.* 2.

1884 SMITH, R. PERCY, M.D., 36, Queen Anne Street, Cavendish Square, W. (C. 1898– .)

1893 SMITH, SOLOMON CHARLES, M.D., Four Oaks, Walton-on-Thames.

O.M. SMITH, Sir THOMAS, Bart., 5, Stratford Place, W. (C. 1869–71, V.P. 1880–2.) *Trans.* 14.

1894 SMITH, THOMAS RUDOLPH, M.B., B.C., 25, Bridge Road, Stockton-on-Tees.

1893 SNAPE, ERNEST ALFRED, M.D., 41, Welbeck Street, W.

1868 SNOW, WILLIAM V., M.D., Richmond Gardens, Bournemouth.

1890 SOLLY, ERNEST, M.B., Strathlea, Harrogate, Yorks. *C.C.* 1.

1888 SPENCER, WALTER GEORGE, M.S., M.B., 35, Brook Street, W. (C. 1896–9.) *Trans.* 9, *C.C.* 8.

1885 SPICER, FREDERICK, M.D., 57, Devonshire Street, W.

1888 SPICER, ROBERT HENRY SCANES, M.D., 28, Welbeck Street, W. *C.C.* 1.

1882 SPOONER, FREDERICK HENRY, M.D., 4, Maitland Place, Lower Clapton, N.E.

1896 SPURRELL, CHARLES, Medical Superintendent, Poplar and Stepney Sick Asylum, Devon's Road, Bromley-by-Bow, E.

1876 SQUIRE, A. BALMANNO, 24, Weymouth Street, W. *Trans.* 5, *C.C.* 4.

1892 STABB, EWEN CARTHEW, 57, Queen Anne Street, W. *C.C.* 1.

1879 STAPLES, FRANCIS PATRICK, Brigade-Surgeon, Army (retired). (Address uncommunicated.)

1896 STEWARD, FRANCIS JAMES, M.B., B.S., 24, St. Thomas's Street, London Bridge, S.E.

1874 Stirling, EDWARD C., M.D. [care of Messrs. ELDER & Co., 7, St. Helen's Place, E.C.], Adelaide, South Australia.

1883 STOKER, GEORGE, 14, Hertford Street, W. *C.C.* 2.

1899 STONE, WILLIAM GREAM, M.B., 93, Denmark Hill, S.E.

ELECTED

1884 STONHAM, CHARLES, 4, Harley Street, W. *C.C.* 3.

1878 STRUGNELL, FREDERICK WILLIAM, Grove End House, Highgate Road, Highgate, N.W. *C.C.* 1.

1878 **Sturge**, WILLIAM ALLEN, M.D., 29, Boulevard Dubouchage, Nice, France.

1894 SUTHERLAND, GEORGE A., M.D., 73, Wimpole Street, Cavendish Square, W. *Trans.* 1.

1872 **SUTHERLAND**, HENRY, M.D., 21, New Cavendish Street, Portland Place, W. *Trans.* 1.

1882 SYMONDS, CHARTERS JAMES, M.S., (*Hon. Secretary*), 58, Portland Place, W. (C. 1888–9, S. 1898– .) *Trans.* 12, *C.C.* 10.

1876 SYMONDS, HORATIO PERCY, 35, Beaumont Street, Oxford.

1885 TAIT, EDWARD SABINE, M.D., 48, Highbury Park, N.

1885 TAIT, HENRY BREWER, Lincluden, Sunnyside Road, Hornsey Lane, N.

1891 TATE, WALTER WILLIAM HUNT, 57, Queen Anne Street, W.

1886 TAY, WAREN, 4, Finsbury Square, E.C.

1878 TAYLER, FRANCIS THOMAS, M.B., 224, Lewisham High Road, S.E.

1897 TAYLOR, E. CLAUDE, M.D., B.S., Eland House, Rosslyn Hill, Hampstead, N.W.

1875 TAYLOR, FREDERICK, M.D., 20, Wimpole Street, W. (S. 1879–81, C. 1882–4, V.P. 1894–7.) *Trans.* 13, *C.C.* 3.

1889 **Taylor**, HENRY HERBERT, 10, Brunswick Place, Brighton.

1890 TAYLOR, JAMES, M.D., 49, Welbeck Street, W. *C.C.* 3.

1882 TAYLOR, SEYMOUR, M.D., 16, Seymour Street, W. *Trans.* 1, *C.C.* 1.

1885 **Taylor**, W. C. EVERLEY, 34, Queen Street, Scarborough.

1886 TEALE, THOMAS PRIDGIN, M.B., F.R.S., 38, Cookridge Street, Leeds. (C. 1897–1900.)

1896 TEMPLETON, GEORGE, M.B., 10, Seymour Street, Portman Square, W.

1890 THANE, EDGAR HERBERT, M.D., Wagga-Wagga, New South Wales.

1882 THIN, GEORGE, M.D., 63, Harley Street, W. *Trans.* 1.

1886 THOMPSON, CHARLES HERBERT, M.D., 17, New Cavendish Street, Portland Place, W.

O.M. THOMPSON, EDMUND SYMES, M.D., 33, Cavendish Square, W. (C. 1880–2.) *Trans.* 1.

O.M. THOMPSON, Sir HENRY, 35, Wimpole Street, W. (C. 1867–8, V.P. 1886–7.) *Trans.* 3.

1897 THOMSON, H. CAMPBELL, M.D., 34, Queen Anne Street, Cavendish Square, W. *C.C.* 1.

1894 THOMSON, STCLAIR, M.D., 28, Queen Anne Street, W. *Trans.* 1, *C.C.* 5.

1896 THORNE, WILLIAM BEZLY, 53, Upper Brook Street, W. *C.C.* 2.

1887 THORNTON, JOHN KNOWSLEY, M.B., C.M., Hildersham Hall, Cambridge. (C. 1890–1.)

1872 THORNTON, WILLIAM PUGIN, 35, St. George's Road, Canterbury. *Trans.* 5.

ELECTED

1885 THURSFIELD, THOMAS WILLIAM, M.D., Selwood, Beauchamp Square, Leamington.

1899 TILLEY, HERBERT, M.D., 101, Harley Street, Cavendish Square, W.

1891 TOMSON, W. BOLTON, M.D., Park Street West, Luton, Bedfordshire.

1897 TOOGOOD, F. SHERMAN, M.D., The Infirmary, 282, High Street, Lewisham, S.E.

1892 TOOTH, HOWARD HENRY, M.D., 34, Harley Street, W.

1887 TOTSUKA, KANKAI, Tokio, Japan.

1874 TRAVERS, WILLIAM, M.D., 2, Phillimore Gardens, Kensington, W.

1884 TREVES, FREDERICK, 6, Wimpole Street, W. (C. 1893.) *Trans.* 7.

1897 TUBBY, ALFRED HERBERT, M.S., 25, Weymouth Street, Portland Place, W. *Trans.* 2, *C.C.* 2.

1899 TULLIS, GEORGE AUGUSTUS, M.D., 20, Clarence Parade, Southsea.

1882 TURNER, GEORGE ROBERTSON, 41, Half Moon Street, W. (C. 1897–1900.) *Trans.* 8. *C.C.* 3.

1888 TURNER, PHILIP DYMOCK, M.D., Claverton House, Ryde, Isle of Wight. *Trans.* 1.

1898 TURNER, WILLIAM, M.B., 53, Queen Anne Street, Cavendish Square, W.

1893 TURNEY, HORACE GEORGE, M.D., 68, Portland Place, W. *C.C.* 4.

1878 TYSON, WILLIAM JOSEPH, M.D., 10, Langhorne Gardens, Folkestone. (C. 1886–8, V.-P. 1900– .) *Trans.* 6.

1881 UHTHOFF, JOHN CALDWELL, M.D., Wavertree House, Brunswick Place, Brighton. (C. 1898– .)

1868 VENNING, EDGCOMBE, 30, Cadogan Place, S.W. (C. 1876–8.) *Trans.* 2.

1890 VOELCKER, ARTHUR FRANCIS, M.D., B.S., 31, Harley Street, W. (C.1900– .) *Trans.* 2, *C.C.* 2.

1886 WADE, CHARLES H., Greenway, Chelston, Torquay.

1868 WAGSTAFFE, WILLIAM WARWICK, Purleigh, St. John's Hill, Sevenoaks. (C. 1878.)

1886 **WAINEWRIGHT**, BENJAMIN, M.B., C.M., 47, Weymouth Street, W. *Trans.* 2, *C.C.* 2.

1885 WAKLEY, THOMAS, jun., 5, Queen's Gate, S.W.

1885 WALKER, CHARLES ROTHERHAM, M.D., 15, Sackville Road, Hove, Sussex.

1899 WALKER, HENRY ROE, 23, Bryanston Street, W.

1896 WALLACE, CUTHBERT SYDNEY, St. Thomas's Hospital, S.E. *Trans.* 3, *C.C.* 2.

1890 WALLIS, FREDERICK CHARLES, M.B., B.C., 26, Welbeck Street, W. *Trans.* 3, *C.C.* 7.

1875 WALSHAM, WILLIAM J., 77, Harley Street, W. (C. 1882–4, V.P. 1896–9.) *Trans.* 5, *C.C.* 1.

1888 WALTERS, FREDERICK RUFENACHT, M.D., 21, Wimpole Street, W. *C.C.* 2.

1896 WARD, EDWARD, M.B., 22, Park Place, Leeds. *Trans.* 1.

1888 Warner, PERCY, Rydal, Woodford Green, Essex.

1894 WASHBOURN, JOHN WICHENFORD, M.D., 6, Cavendish Place, Cavendish Square, W. *Trans.* 3, *C.C.* 1.

ELECTED

1891 WATERHOUSE, HERBERT FURNIVALL, M.D., C.M., 81, Wimpole Street, W.

1896 **WEBER**, FRED. PARKES, M.D., 19, Harley Street, W. *C.C.* 5.

O.M. WEBER, Sir HERMANN D., M.D., 10, Grosvenor Street, W. (C. 1867–71, V.P. 1873–5.) *Trans.* 9.

1882 WEST, SAMUEL, M.D., 15, Wimpole Street, W. (C. 1887–9.) *Trans.* 16, *C.C.* 5.

1895 WETHERED, FRANK J., M.D., 83, Harley Street, W.

1874 WHEELHOUSE, CLAUDIUS GALEN, Hilary Place, Leeds. *Trans.* 1.

1868 WHIPHAM, THOMAS TILLYER, M.D., 11, Grosvenor Street, W. (C. 1878–80, V.P. 1894–7.) *Trans.* 13.

1891 WHITE, CHARLES PERCIVAL, M.B., B.C., 22, Cadogan Gardens, S.W.

1882 WHITE, EDWIN FRANCIS, Westlands, 280, Upper Richmond Road, Putney, S.W.

1890 WHITE, GILBERT B. MOWER, M.B., B.S., 112, Harley Street, W.

1883 WHITE, WILLIAM HALE, M.D. 65, Harley Street, W. (C. 1887–9, 1896–9, S. June, 1893–6.) *Trans.* 14, *C.C.* 4.

1883 WHITE, WILLIAM HENRY, M.D., 43, Weymouth Street, W. *Trans.* 1. *C.C.* 1.

1894 WHITELOCKE, RICHARD HENRY ANGLIN, M.B., C.M., 6, Banbury Road, Oxford.

1897 WHITFIELD, ARTHUR, M.D., 12, Upper Berkeley Street, Portman Square, W.

1897 WHITING, ARTHUR JOHN, M.D., 142, Harley Street, W.

1882 WHITTLE, EDWARD GEORGE, M.D., 65, Dyke Road, Brighton.

1871 WIGHT, GEORGE, M.B., C.M., 428, Liverpool Road, N.

1879 WILCOX, HENRY, M.B., Newlyn, Fleet, Hants.

1894 **WILKIN**, GRIFFITH CHARLES, West Coker, near Yeovil, Somerset.

1884 WILLCOCKS, FREDERICK, M.D., 14, Mandeville Place, W. *C.C.* 1.

O.M. WILLETT, ALFRED, 36, Wimpole Street, W. (C. 1872–5, V.P. 1889–91.) *C.C.* 2.

1890 WILLETT, EDGAR, M.B., 25, Welbeck Street, W.

1898 WILLEY, FREDERICK INGOR, M.B., B.S., The Wych, 8, Avenue Road, Highgate, N.

1888 WILLIAMS, CAMPBELL, 18, Queen Anne Street, Cavendish Square, W. *Trans.* 1.

O.M. WILLIAMS, CHARLES THEODORE, M.D., 2, Upper Brook Street, W. (C. 1877–9, V.P. 1889–90.) *Trans.* 8.

1888 WILLIAMS, DAWSON, M.D., 101, Harley Street, W. (C. 1893–7.)

1881 WILLIAMS, Sir JOHN, Bart., M.D., 63, Brook Street, W. (C. 1885–6.)

1890 WILLIAMS, W. ROGER, Beaufort House, Clifton Down, Clifton, Bristol.

1876 WILLIAMSON, JAMES MANN, M.D., Ventnor, Isle of Wight.

O.M. WILLIS, FRANCIS, M.D., The Spa, Braceborough, Stamford.

1893 WILLS, JOSEPH PEARCE BUDGETT, M.D., Bexhill, Hastings.

1889 WILLS, WILLIAM ALFRED, M.D., 29, Lower Seymour Street, W.

ELECTED

1886 WILSON, ALBERT, M.D., Minto House, Fairlop Road, Leytonstone, Essex. *Trans.* 1, *C.C.* 2.

1888 Wilson, CLAUDE, M.D., C.M., Belmont, Tunbridge Wells. *Trans.* 2.

1891 WILSON, THEODORE STACEY, M.B., C.M., 65, Temple Row, Birmingham.

1890 WOOD, NEVILLE, 42, Elvaston Place, Queen's Gate, S.W.

1883 WOODCOCK, JOHN ROSTRON, Abberley, near Stourport, Worcestershire.

1879 WOODWARD, GEORGE P. M., M.D., Deputy Surgeon-General; Sydney, New South Wales.

1894 WOOLLETT, CHARLES JEROME, "Ambleside," Streatham, S.W.

1884 WORTS, EDWIN, 6, Trinity Street, Colchester.

1888 WYMAN, WILLIAM S., M.D., Red Brae, 18, Putney Hill, S.W.

1892 WYNTER, WALTER ESSEX, M.D., B.S., 30, Upper Berkeley Street, W. *C.C.* 2.

1872 YEO, I. BURNEY, M.D., 44, Hertford Street, W. (C. 1881–3.) *Trans.* 8.

[*It is requested that any change of Title or Residence be communicated to the Secretaries before the* 1*st of July in each year, in order that the list may be made as correct as possible.*]

LIST OF MEMBERS.

ORIGINAL MEMBERS (ALPHABETICALLY).

Sir Henry Acland, Bart., M.D., F.R.S.
Henry Arnott.
Richard Barwell.
Henry Charlton Bastian, M.D., F.R.S.
Sir Wm. Henry Broadbent, Bart., M.D., F.R.S.
Thomas Bryant.
Thomas Buzzard, M.D.
William Cayley, M.D.
William Selby Church, M.D.
Edward Clapton, M.D.
John Couper.
John Croft.
William Howship Dickinson, M.D.
Sir Dyce Duckworth, M.D.
Christopher Heath.
Timothy Holmes.
Carsten Holthouse.
Jonathan Hutchinson, F.R.S.
J. Hughlings Jackson, M.D., F.R.S.
Charles Kelly, M.D.
John Langton.
George Lawson.
Arthur Trehern Norton.
Thomas William Nunn.
John William Ogle, M.D.
Frederick William Pavy, M.D., F.R.S.
Thomas Pickering Pick.
Sir Richard Douglas Powell, Bart., M.D.
Sydney Ringer, M.D., F.R.S.
Sir Thomas Smith, Bart.
Edmund Symes Thompson, M.D.
Sir Henry Thompson.
Sir Hermann D. Weber, M.D.
Alfred Willett.
Charles Theodore Williams, M.D.
Francis Willis, M.D.

ARRANGED ACCORDING TO DATE OF ELECTION.

1868 Constantine Holman, M.D.
Thomas Tillyer Whipham, M.D.
Christian G. H. Bäumler, M.D.
James Grey Glover, M.D.
T. Henry Green, M.D.
Howard Marsh.
Charles Prentis.
Edgcombe Venning.

1868 John Ford Anderson, M.D.
George Granville Bantock, M.D.
George Charles Bright, M.D.
Frank W. Cooper.
Julian Evans, M.B.
Edward Hooper May, M.D.
William Warwick Wagstaffe.
William Ogle, M.D.

1868 James Edward Pollock, M.D.
Franz Oppert, M.D.
William V. Snow, M.D.
Charles Drage, M.D.
Frederick Royston Fairbank, M.D.
Michael W. Rice, M.D.
Louis Stromeyer Little.

1869 Leonard William Sedgwick, M.D.
J. Warrington Haward.
Henry Frederick Augustus Goodridge, M.D.
Olliver Thomas Duke, M.B.

1871 Julius Althaus, M.D.
Sir William Mac Cormac, Bart., K.C.V.O.
Alfred Playne, M.B.
George Wight, M.B.
Ebenezer Diver, M.D.
George Vivian Poore, M.D.

1872 I. Burney Yeo, M.D.
Henry Harris, M.D.
William Pugin Thornton.
G. Anderson Critchett.
J. C. J. Fenwick, M.D.
Andrew J. Myrtle, M.D.
Sir William Bartlett Dalby.
Thomas Ridge Jones, M.D.
George Slight, M.D.
Henry Sutherland, M.D.

1873 William Julius Mickle, M.D.
Robert William Parker.
David Lloyd Roberts, M.D.
George Henry Savage, M.D.
Edwin Chisholm, M.D.
Thomas Churton, M.D.

1874 John Hammond Morgan.
Edward R. Rowland.
Claudius Galen Wheelhouse.
Edward C. Stirling, M.D.
William Henry Bennett.
William Travers, M.D.
William Brown Holderness.
Andrew Clark.

1875 Thomas Barlow, M.D.
Sidney Coupland, M.D.
Clinton T. Dent.
C. D. B. Hale.
Edward Liveing, M.D.
EdwardNettleship.
William J. Walsham.
Rickman John Godlee, M.S.
Arthur Paul Sherwood.
T. Gilbart-Smith, M.D.

1875 James Frederic Goodhart, M.D.
Sir William Richard Gowers, M.D., F.R.S.
William Smith Greenfield, M.D.
Shirley F. Murphy.
Herbert W. Page.
Frederick Taylor, M.D.

1876 Arthur E. J. Barker.
Horatio Percy Symonds.
A. Balmanno Squire.
David White Finlay, M.D.
Henry Greenway Howse, M.S.
Furneaux Jordan.
R. Clement Lucas, B.S.
James Mann Williamson, M.D.
George Buckston Browne.
Arthur Edwin Temple Longhurst, M.D.

1877 Robert Hogarth Clay, M.D.
A. Pearce Gould, M.S.
Henry Radcliffe Crocker, M.D.
David B. Lees, M.D.
Walter Hamilton Acland Jacobson, M.B., M.Ch.
Isambard Owen, M.D.
William Ewart, M.D.
Henry Morris, M.B.
William Miller Ord, M.D.
Henry Ambrose Lediard, M.D.
Bernard Roth.
Henry Hugh Clutton.
Malcolm Alex. Morris.

1878 George P. Field.
Thomas Warner Lacey.
Thomas Colcott Fox, M.B.
Sir Felix Semon, M.D.
Henry de Fonmartin, M.D.
C. H. Golding-Bird, M.B.
Donald William Charles Hood, M.D.
Lord Lister, F.R.S.
Francis Thomas Tayler, M.B.
F. de Havilland Hall, M.D.
William Allen Sturge, M.D.
William Joseph Tyson, M.D.
William Johnston, M.D.
Charles Robert Bell Keetley.
William Appleton Meredith, C.M.

1879 Frederick William Strugnell.
William Edward Burton.
James Magill, M.D.
Edward Markham Skerritt, M.D.
Henry Wilcox, M.B.
John Abercrombie, M.D.

1879 F. G. Dawtrey Drewitt, M.D.
Stephen Mackenzie, M.D.
William Harrison Cripps.
Francis Patrick Staples.
Geo. Courteney Henderson, M.D.
Thomas John Maclagan, M.D.
Henry Davy.
Thos. Walter Harropp Garstang.
George Lichtenberg, M.D.
Charles W. Mansell Moullin.
John Reuben Lunn.
George P. M. Woodward, M.D.
Robert Peel.
Frederic S. Dennis, M.D.
1880 T. Mark Hovell.
Wyndham Cottle, M.D.
Henry Francis Baker.
Bernard O'Connor, M.D.
Charles Edward Beevor, M.D.
1881 George Henry Makins.
Robert William Burnet, M.D.
James Kingston Fowler, M.D.
Charles Edward Harrison, M.B.
Malcolm Macdonald McHardy.
Rushton Parker.
Sir John Williams, Bart, M.D.
Montagu Lubbock, M.D.
William Pasteur, M.D.
John Caldwell Uhthoff, M.D.
Henry Treutham Butlin.
H. A. Powell, M.A.
1882 George Robertson Turner.
E. Noble Smith.
Robert William Goldie.
Frederick Charles Barker, M.D.
William Henry Kesteven.
Frederic Morell Mackenzie.
Daniel Colquhoun, M.D.
Seymour Taylor, M.D.
Philip Henry Bindley, M.B.
Edward George Whittle, M.D.
D. H. Goodsall.
Frederick Henry Spooner, M.D.
Frederick Haycraft Berry, M.D.
Herbert Collier, M.D.
Samuel West, M.D.
Emile Emond, M.D.
Charters James Symonds, M.S.
Angel Money, M.D.
C. F. Coxwell, M.B.
George Allan Heron, M.D.
Augustus Joseph Pepper, M.B.
Harrington Sainsbury, M.D.
George Thin, M.D.
1882 Edwin Francis White.
1883 Anthony A. Bowlby.
Percy Kidd, M.D.
William Henry White, M.D.
Hubert Montague Murray, M.D.
Robert Fitzroy Benham.
William Henry Allchin, M.D.
John Mitchell Bruce, M.D.
William Arbuthnot Lane, M.S.
Bernard Pitts.
William Hale White, M.D.
William Coode Adams, M.B.
William Anderson.
Robert Leamon Bowles, M.D.
George Henry Jackson.
George Hunt Orton, M.B.
John Liston Paul, M.D.
Thomas Laurence Read.
Frederick Thomas Roberts, M.D.
Charles Alfred Ballance, M.S.
John Rostron Woodcock.
1884 Frederick Willcocks, M.D.
R. Percy Smith, M.D.
Edgar Duke.
John Mackern, M.B.
Paul M. Chapman, M.D.
Wilmot Parker Herringham, M.D.
Philip Henry Pye-Smith, M.D. F.R.S.
Charles Stonham.
Dudley Wilmot Buxton, M.D.
Edwin Worts.
Seymour J. Sharkey, M.D.
Frederick Treves.
John James Pringle, M.B.
Frederick Lucas Benham, M.D.
Walter Edmunds, M.C.
Stephen Paget.
Lambert Hepenstal Ormsby, M.D.
John Poland.
Edwin Leonard Adeney, M.D.
Victor Horsley, F.R.S.
Henry Carr Maudsley, M.D.
Bilton Pollard, B.S.
1885 Frederick Spicer, M.B.
Herbert Larder.
James Berry, B.S.
Fred. Walker Mott, M.D., F.R.S.
George Newton Pitt, M.D.
W. C. Everley Taylor.
Sidney Phillips, M.D.
A. W. Mayo Robson.
Thomas Wakley, jun.
Herbert William Allingham.

1885 Thomas William Thursfield, M.D.
Alexander Dalton Murray, M.B.
Robert Maguire, M.D.
Robert Alexander Gibbons, M.D.
Henry Brewer Tait.
Charles Rotherham Walker, M.D.
Richard Caton, M.D.
Arthur Henry Robinson, M.D.
Edward Sabine Tait, M.B.
William Bruce Clarke, M.B.
Charles Barrett Lockwood.
Reginald J. Ryle, M.D.
J. Michell Clarke, M.B.
Henry George Armstrong.
Roderick Maclaren, M.D.
W. Watson Cheyne, F.R.S.
Edward Liveing Fenn, M.D.

1886 Thomas Dixon Savill, M.D.
John Cahill.
Charles Henry Wade.
Benjamin Wainewright, M.B.
Waren Tay.
William John Penny.
William Henry Battle.
James Hardie, M.D.
Francis Henry Hawkins, M.D.
R. Hingston Fox, M.D.
Henry Edward Juler.
John Ward Cousins, M.D.
Joseph Frank Payne, M.D.
T. Pridgin Teale, F.R.S.
H. Lankester, M.D.
Arthur T. Davies, M.D.
Charles Herbert Thompson, M.D.
Arthur Quarry Silcock, M.D., B.S.
Henry Handford, M.D.
Alfred Scott.
Albert Wilson, M.D.

1887 Archibald E. Garrod, M.D.
H. T. Rutherfoord, M.B.
Kankai Totsuka.
Thomas Oliver, M.D.
Francis George Penrose, M.D.
Samuel Herbert Habershon, M.D.
John Knowsley Thornton.
John Bland-Sutton.
Oswald Auchinleck Browne, M.D.
Joseph Arderne Ormerod, M.D.
J. H. E. Brock, M.B., B.S.
Francis William Clark.
A. H. Weiss Clemow, M.D., C.M.
E. Hurry Fenwick.
R. Lawford Knaggs, B.C.
John D. Malcolm, M.B., C.M.

1887 Sidney Martin, M.D., B.S., F.R.S.
Thomas Horrocks Openshaw, M.B.

1888 A. G. Barrs, M.D.
J. W. Batterham, M.B., B.S.
Montagu Handfield-Jones, M.D.
Alfred Rice Oxley, M.D.
Arthur Roper, M.D.
Robert Henry Scanes Spicer, M.D.
Campbell Williams.
Frederic S. Eve.
Alexander Morison, M.D.
Frederick Page, M.D.
Frederick J. Smith, M.D.
Frederick R. Walters, M.D.
Claude Wilson, M.D., C.M.
Charles H. Gage-Brown, M.D.
Arthur Jamison, M.D., C.M.
J. H. Menzies.
Frank Ernest Roberts.
George Stoker.
Robert Ashton Bostock.
Hugh Armstrong.
Hyde Marriott, M.B.
Percy Warner.
J. T. James, M.D.
Edwin A. Barton.
W. Page May, M.D.
Philip D. Turner, M.D.
William S. Wyman, M.D.
Dawson Williams, M.D.
Augustus W. Addinsell, M.B., C.M.
John Anderson, M.D.
Henry French Banham, M.D.
George Haynes Hetherington.
David James Mason, M.D., C.M.
Walter G. Spencer, M.B., M.S.

1889 Theodore Dyke Acland, M.D.
Raymond Johnson, M.B., B.S.
H. Davy Rolleston, M.A., M.D.
P. MacBride, M.D.
D. Newman, M.D.
Herbert Elwin Harris, M.B.
John E. Ranking, M.D.
William Alfred Wills, M.D.
Edward Ashby Fardon.
Stanley Boyd, M.B.
George Ezra Halstead, M.D., B.S.
Henry Herbert Taylor.
John Duncan, M.D.
Wm. Wallis Ord, M.D., B.Ch.
Leonard Arthur Bidwell.
Arthur J. M. Bentley, M.D.

1889 Francis R. B. Bisshopp, M.B.
Henry Percy Dean, M.B.
Louis Albert Dunn, M.S.
Daniel Mackay Forbes.
H. Pennell Hawkins, M.D., B.Ch.
Lauriston Elgie Shaw, M.D.
1890 John Rose Bradford, M.D., F.R.S.
J. Fletcher Little, M.B.
Robert Robertson, M.D.
Ernest Solly, M.B.
James Taylor, M.D.
Francis O. Buckland, B.A., M.B., C.M.
E. Baxter Forman, M.D.
G. Somerville Robinson.
Edmund W. Roughton, B.S.
Edgar Willett, M.B.
Thomas H. Rickard Crowle.
James Calvert, M.D.
H. Roxburgh Fuller, M.D.
Arthur F. Voelcker, M.D.
Neville Wood.
W. Roger Williams.
Gilbert B. M. White, M.B., B.S.
Frederick Charles Wallis, M.B., B.C.
Alfred Parkin, M.S.
George A. Hawkins-Ambler.
James Harper, M.D.
Walter Henry Brown.
John Walter Carr, M.D.
Ernest Le Cronier Lancaster, M.B., B.Ch.
Patrick Manson, M.D., C.M.
Miles Miley, M.A., M.B.
Edgar Herbert Thane, M.B.
Charles William Chapman, M.D.
Michael G. Foster, M.A., M.B.
1891 Frederic François Burghard, M.D., M.S.
Roger Neville Goodman, M.B.
Herbert Furnivall Waterhouse, M.D., C.M.
Walter William Hunt Tate.
William Lee Dickinson, M.D.
Greville MacDonald, M.D.
J. Kingston Barton.
J. Dundas Grant, M.D.
W. Kington Fyffe, M.B., B.C.
Albert Carless, M.B., M.S.
W. Bolton Tomson, M.D.
Harry Littlewood, M.D.
1891 Hector W. G. Mackenzie, M.A., M.D.
Chas. Percival White, M.B., B.C.
Arnold Caddy.
Theodore Stacey Wilson, M.B., C.M.
1892 William Hunter, M.D.
Frank Thomas Paul.
Walter Essex Wynter, M.D., B.S.
Damer Harrison.
John Alfred Masters, M.D.
Walter Giffard Nash.
John Stephenson Selwyn-Harvey, M.D.
Ewen Carthew Stabb.
Edward John Lewis, M.B., B.C.
Henry Betham Robinson, M.D. M.S.
Richard James Herbert Scott.
Howard Henry Tooth, M.D.
1893 John Ernest Paul, M.B.
James William Bond, M.D.
Harry Campbell, M.D.
W. Soltau Fenwick, M.D.
Ernest Alfred Snape, M.D.
Lewis G. Glover, M.B., B.C.
Solomon Charles Smith, M.D.
William Bracewell Mercer, M.B., B.C.
Robert Henry Cole, M.D.
Donald Rose Paterson, M.D., C.M.
Walter Spencer Anderson Griffith, M.D.
Alfred Theodore Rake, M.B., B.S.
Francis Charles Abbott, M.B., B.S.
James William Browne, M.B.
Edwin Harding Lendon, M.B.
George Redmayne Murray, M.D., M.A.
R. J. Bliss Howard, M.D.
Henry Jones Curtis, M.D., B.S.
Henry Albert Caley, M.D.
John Henry Dauber, M.B.
Theodore Henry Ionides, M.B., B.S.
Arthur Foxwell, M.D.
Horace George Turney, M.D.
Henry Grabham Lys, M.D.
Arthur Ernest Sansom, M.D.
John Henry Bryant, M.D.
G. Johnston, M.B.
Willmott H. Evans.
Charles Gibbs.
Robert Stephen Charsley.

1893 Joseph Pearce Budgett Wills, M.D.
Arnold Lawson, M.D.
1894 Herbert Morley Fletcher, M.D.
John Wichenford Washbourn, M.D.
Edward Wilberforce Goodall, M.D.
Harry Gilbert Barling.
Ernst Michels, M.D.
Charles Jerome Woollett.
StClair Thomson, M.D.
George A. Sutherland, M.D.
John Murray.
Richard Henry Anglin Whitelocke, M.B., C.M.
John Attlee, M.D., B.C.
Griffith Charles Wilkin.
Thomas Hugh Dickson, M.B.,B.C.
C. Gordon Brodie.
Bernard Scott.
John Porter Parkinson, M.D.
Thomas Rudolph Smith, M.B., B.C.
Arthur Pearson Luff, M.D.
1895 Herbert Murray Ramsay.
Arthur G. Phear, M.D., B.C.
Ernest E. Lewis, M.D.
Karl Grube, M.D.
Gerald R. Baldwin.
James Galloway, M.D.
F. B. Willmer Phillips, M.B.
Charles Henry Hough.
Leonard George Guthrie, M.D.
James Ernest Lane.
Frank J. Wethered, M.D.
1896 Richard Ackerley.
Charles Spurrell.
Cuthbert Sidney Wallace.
Charles R. Box, M.D.
Walter Stacy Colman, M.D.
Edward Ward, M.B.
Norman Dalton, M.D.
Fred. Parkes Weber, M.D.
Francis James Steward, M.B., B.S.
William Bezly Thorne, M.D.
Douglas Drew, M.D., B.S.
Surg.-Capt. W. R. Crooke-Lawless, A.M.S., M.D.
William Henry Crosse.
William Elliot Risdon, M.D.
Robert Milne Beaton, M.B.
Priestley Leech, M.D.
1896 Ebenezer Stanley Smith, M.D.
G. H. A. Comyns Berkeley, M.B.
Edward Cooper Bentham.
Robert Herbert Mills-Roberts.
Jonathan Hutchinson, jun.
Frederick E. Batten, M.D.
Farquhar Mac Rae, M.B.
Rutherford John Pye-Smith.
George Templeton.
Bertram Louis Abrahams, M.B., B.Sc.
1897 Raymond H. P. Crawfurd, M.D.
John Henry Deane.
Percy Furnivall.
Arthur Whitfield, M.D.
Frederick William Forbes Ross, M.D.
Harold Low, M.B.
Charles Ralph Keyser.
George Bertram Hunt, M.B.
Hugh Roger-Smith, M.D.
Alfred Herbert Tubby, M.S.
Charles Lamplough.
H. Campbell Thomson, M.D.
Alfred P. Hillier, M.D., C.M.
F. Sherman Toogood, M.D.
E. Claude Taylor, M.D.
Charles Humphrys.
Charles Sempill de Segundo, M.B.
Arthur John Whiting, M.D.
Alfred Downing Fripp, M.S.
Chichester Gould May, M.D.
1898 J. J. Perkins, M.D.
James Jackson Clarke, M.B.
Ernest Herbert Cobb.
Sydney Backhouse Hulke.
Thomas Herbert Kellock.
William Darley-Hartley.
Robert Jones.
Arthur Philip Beddard, M.B.
Alfred Ernest Russell, M.D., B.S.
William Cecil Bosanquet, M.D.
Frederick Ingor Willey, M.B.
Jonathan F. C. H. Macready.
Arthur A. H. Partridge, M.B., M.Ch.
Leonard Avery.
Herbert Challice Crouch.
William Turner, M.B., B.S.
Philip Frank, M.D.
Harold Batty Shaw, M.D.

1898 Joseph Stewart Richards, M.D., B.S.
William Henry Breffit Brook, M.D., B.S.
1899 George Edward Rennie, M.D.
W. Warren Low, M.D., B.S.
Rayner Derry Batten, M.D., B.S.
Charles E. Forbes Mouat-Biggs.
Rutherford Morison.
Murray Leslie, M.D.
Herbert Tilley, M.D.
Charles Owen Fowler, M.D.
Cyril Ogle, M.B.
Charles Herbert Fagge, M.S.
Claude Somerville Hawkes.
Henry Stephen Sandifer, M.D., B.S.
Marcus Marwood Bowlan, M.B.

1899 Alfred Eid, M.D.
Karl Fürth, M.D.
Wm. Gream Stone, M.B.
Andrew Duncan, M.D.
Edmond Lucien De Chazal, M.D.
Henry Roe Walker.
Gustave Schorstein, M.B.
Edwards Collingwood Andrews, M.D.
1900 Berkeley George Andrew Moynihan, M.S.
Harry Cooper, M.D.
George Augustus Tullis, M.D.
Griffith Lloyd Roberts, M.B., C.M.
Edward Percy Paton, M.S.
Kenneth Scott, M.B.
George Steele Perkins, M.D.
John Arthur Hayward, M.D.

REPORT

OF THE

COUNCIL OF THE CLINICAL SOCIETY,

MAY, 1900.

THE COUNCIL is glad to be able to report that during the past Session the activity and usefulness of the Society have in no way diminished. Numerous papers of importance have been read and discussed, and the Clinical Evenings have continued to be well attended.

Sixteen Ordinary Members and one Honorary Member—Mr. Christopher Heath—have been elected, the total number of Ordinary Members being 582.

The Society has lost by death Sir James Paget, Bart., an Original and Honorary Member, and President in 1869-70; Mr. B. E. Brodhurst and Dr. R. Southey, both Original Members; Mr. J. Neville Davies-Colley and Dr. F. Charlewood Turner; while five Ordinary Members have resigned.

The Treasurer's Statement shows the financial position of the Society to be satisfactory.

THE CLINICAL SOCIETY OF LONDON.

Statement of Cash Receipts and Payments from the 1st May, 1899, to the 30th April, 1900.

HOWARD MARSH, Esq., *Treasurer.*

RECEIPTS.	£	s.	d.
Balance, 1st May, 1899	224	4	11
173 Subscriptions received by Collector . £181 13 0			
201 „ „ Bank . 211 1 0			
374	392	14	0
20 Admission Fees at £2 2s.	42	0	0
1 Composition Fee (*Transactions*)	5	5	0
Sale of *Transactions*	19	2	10
Sale of Antitoxin Report		5	3
Dividend on Consols (£600)	15	19	0
	£699	11	0

PAYMENTS.	£	s.	d.
Cost of *Transactions*, Vol. XXXII:			
Printing, paper, binding, delivery, &c. . £161 14 4			
Illustrations . 74 8 0			
	236	2	4
Meetings:			
Rent of Rooms . £105 0 0			
A. Tapson (attendance) . 7 10 0			
Refreshments . 33 8 0			
	145	18	0
Treasurer and Hon. Secretaries:			
Secretarial assistance . £26 5 0			
Commission to Collector . 15 15 0			
	42	0	0
Stationery and Printing . £38 16 0			
Hire of Microscopes and Lantern . 3 13 0			
	42	9	0
Petty expenditure	8	14	4
Balances:			
At Bank . £223 13 1			
Petty Cash . 14 3			
	224	7	4
	£699	11	0

R. DOUGLAS POWELL, *President.*
HOWARD MARSH, *Treasurer.*
CHARTERS J. SYMONDS, *Hon. Secretary.*

7th May, 1900.

Examined with the vouchers and found correct.

CHAS. W. CHAPMAN,
CHARLES SPURRELL, } *Auditors.*

COMMUNICATIONS.

I.—*Aneurysm of the first part of the Arch of the Aorta projecting into the Right Axilla.* By T. CHURTON, M.D. *Read October* 13, 1899.

ANDREW C., a boatman æt. 53, was admitted into the Leeds Infirmary on June 25, 1898, complaining of a painful swelling on the right side of the chest. The swelling extended from the second to the fourth rib, and from mid-axilla to the nipple line. In size it was about equal to half a cocoa-nut; it had a well-marked expansile pulsation; was not very tender; the skin was not discoloured; the pain was not intense. The pulsation was decidedly greater than is commonly felt in pulsating sarcoma, and was evidently due to an aneurysm. The patient stated that there had existed a sensation of fulness and discomfort in his right chest for two years, and that three months ago shooting pain in his right arm compelled him to give up his work on a barge. One month ago he found a swelling as large as a hen's egg in his right armpit, and this increased in size slowly for a week. It then "suddenly" enlarged, forming a tumour so prominent that the arm could not be brought against the side. For the last fortnight he had been confined to bed; he was very feeble, and had had some "anginal" attacks; there was still a dull pain in the area of the tumour. This history of pressure upon nerves and ribs, with finally yielding of the ribs and consequent rapid extension of the tumour, agreed with the diagnosis of an aneurysm.

An aortic regurgitant murmur was heard at or near the

fourth left costal cartilage. Syphilis was denied, but there was no history of rheumatism; it seemed probable that the bruit might be associated with chronic aortitis, whether syphilitic or not.

As to the origin and exact site of the aneurysm. The patient had been sent to the hospital as a case possibly for operation, and had been there briefly examined by a surgeon of great experience, but merely with a view to his suitability for admission. He thought at a glance, as it were, that it might be of axillary origin. After admission one resident agreed that it was axillary, another believed it to be aortic, the diagnosis turning upon the effect of pressure upon the subclavian artery, which appeared to one of the examiners to partially check the pulsation in the tumour, and to the other to have no effect upon it.

Upon closer investigation a pulsation, though not visible nor obtrusive, could be distinctly—or perhaps it might be more correct to say obscurely—felt across the right chest from the tumour to the sternum. This pulsating or slightly heaving area was dull from the second rib downwards; the heart sounds were plainly heard all over the dull area, no lung sounds being present there, and the heart's impulse was found rather far over to the left, in the sixth space, an inch and three quarters external to the nipple line. This displacement of the impulse could not be entirely due to the valvular defect. The bruit was fine and whiffing or a light blowing, not suggestive of a large regurgitation; nor did the radial pulse, though it was thought to be aortic, indicate a highly incompetent valve.

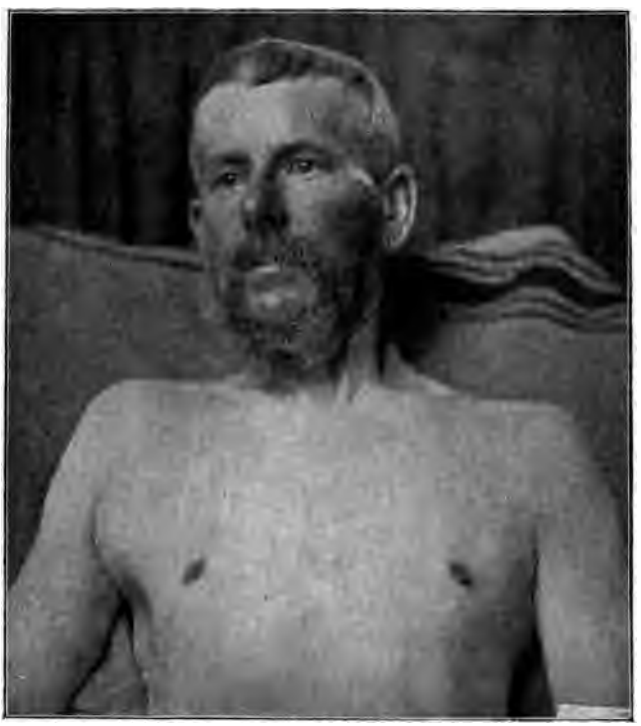
There were no other marked pressure signs. There was not even any distinct anæsthesia in the skin area supplied by the intercostal nerves implicated; the patient was, however, when tested, too languid to appreciate small differences in sensibility. The phrenic nerve was undisturbed; the right half of the diaphragm acted perfectly in deep inspiration. Examination of the lung was difficult and imperfect; there was no cough or other marked symptom: pulse 80, respirations 24. There was no distension of superficial veins, no enlargement of liver, no ascites, and no œdema.

I at first surmised that an aneurysm originating just beyond the pericardial line on the outer side of the aorta had insinuated itself between the upper and middle lobes of the right lung, and in its growth had separated them. But this was not very probable, and, as will be seen in the specimen,

had not happened. No doubt it is easy in some lungs, not in all, to separate these lobes up to the very root without tearing lung tissue, but merely pleura and connective tissue. It is, however, not likely that the pressure of an aneurysm would have any other effect than adhesion and sealing up of the path between the lobes, if any existed.

On July 3 the external swelling again increased in size;

FIG. 1.



on the 5th the patient died with symptoms of internal hæmorrhage.

A *post-mortem* examination was made by my house physician, Mr. A. C. Pierson, who removed the parts *en masse*. *The heart* is enlarged, chiefly in the left ventricle. The mitral valve is normal; aortic cusps are rather thick, especially the lunula, but there is no contraction or marked deformity.

The cavity is much enlarged, but the muscle is thick. *The aorta* for the first inch is fairly good; beyond this point it is greatly dilated, even within the pericardium. Beyond the pericardial attachment the first part of the arch bulges outwards and downwards. On its outer aspect is a long narrow slit admitting four or five fingers—this is just outside the pericardium; the orifice looks outwards and upwards—perhaps backwards also. This opening in the artery leads into a *sac*, which now measures 5½ inches in vertical, 4½ inches in lateral, and 3½ inches in antero-posterior diameter. It has a thick lining of solid clot; within this was a mass of soft black clot. At the upper and inner part of the sac was a small opening three quarters of an inch in length, from which blood had escaped in the pleura at the upper and posterior part of the thorax. The third and fourth ribs appear to have been broken in the anterior axillary region, and the remains of the broken and separated parts are apparently contained in the wall of the sac. It will be seen in the specimen that the ribs from which the cartilage has been removed, along with the sternum, are intact as to their outer surface for two or three inches.

The *aneurysm* lies wholly in front of the lung. The middle or lower part of the inner face of the upper lobe is so firmly adherent to the aneurysm, that it has been torn in the attempt to separate it, leaving a patch of lung tissue upon the posterior wall. This appears to have been the first part of the lung reached and compressed by the aneurysm, which, occuring in a dilated aorta, formed with it a bridge over the root and the lung immediately adjacent. It seems probable that it was owing to the compression of the aneurysm having been first brought to bear on the bronchi leading to the lower part of this lobe, while the other bronchi were left free, that the sac in its extension was able to cause its collapse, to pass over it to the outer part of the thorax, instead of taking the usual course to the front. The finger could readily be passed through the tunnel thus formed, tearing slight adhesions between the dilated aorta in front and the root of the lung behind. The apex of the upper lobe, and the middle and the lower lobes, do not seem to have been greatly affected by the aneurysmal pressure.

The point of projection of the aneurysm seems to be very unusual, and in my limited inquiry among notes, books and persons, it is at present, indeed, unique.

II.—*A case of Obstinate Anæmia in a Young Girl associated with peculiar trophic disturbances, and with doubtful Thrombosis of Intra-cranial Veins.* By Fred. J. Smith, M.A., M.D. *Read October* 13, 1899.

THE history of this case, on which I propose to comment, is as follows:—E. H., æt. 19, was first sent to me in November, 1895. My note made at the time states that she was the most marked case of anæmia I had ever seen. She was one of twins at birth, and was said to have been a very small baby; began to menstruate at the age of twelve and a half, and lost very much at her periods, which lasted over a week each time. She was reported to have had an ulcer of the stomach in 1894, but I have had no evidence of this of a definite character. She is certainly under the average in height, but otherwise a fairly-proportioned girl, and I may here mention that later I had her examined by Dr. Lewers, who reported to me that he could detect no definite want of development in the pelvic organs.

She came into hospital in November, 1895, and improved very much under rest with Blaud's pills and a mixture of Decoct. Aloes Co. and Mist. Ferri Co. āā ℥ss. Shortly after leaving she began to complain of œdema of the legs and a good deal of pain in them, and I feared an affection of the nervous system, but she again rapidly improved and went out in her usual health in the middle of January, 1896, and I saw her in March, 1896, still fairly well.

From that time she has, however, been more or less constantly under my care, and in and out of hospital with the anæmia, and very varied symptoms due no doubt to poverty of blood.

During 1896 she continued to take enormous numbers (as many as twenty-one *per diem*) of Blaud's pills, and seemed to keep fairly well, though never becoming a good colour. In 1897 she was again an in-patient for the anæmia, and also for vague nerve symptoms, pain in the feet and legs, with patchy blunting of sensation, and I had a suspicion that there might be peripheral neuritis. During the year she was treated with iron, arsenic, manganese, bone-marrow, etc.,—in fact,

every drug that I could hear or think of as possibly assisting anæmia.

In and during 1898 she developed warts on the hands, and on her own initiative applied nitric acid to them. This not only destroyed the warts, but left large gaping ulcers which healed but slowly, showing a great lack of trophic power. At the end of April I again admitted her for excruciating headaches. Her admission note reads, "Admitted for severe headache; profound anæmia, getting worse in spite of treatment; spleen not enlarged; liver not enlarged; glands to be felt in axillæ and groins, but not enlarged; fundi anæmic, but no papillitis, retinitis, or hæmorrhages; some slight œdema of forehead."

Her blood was frequently examined in 1898, and showed about 4,000,000 corpuscles with 30 per cent. hæmoglobin, the usual clinical picture of simple anæmia. Quinine, antipyrin, iron, arsenic, bone-marrow, and morphia were all tried to relieve the disease and its symptoms, but my impression was that time had more to do with relief than anything else. She was discharged in June, 1898, however, somewhat improved.

She repeatedly visited me at the hospital during the latter half of 1898 and until April 28, 1899. Her usual complaints were tiredness and pains in the legs, and headache. Nitroglycerine, phenacetin, and antipyrin all seemed to help a little; but none, nor mixtures of them, gave marked relief.

I again admitted her on April 28, 1899, for the commencement of the strangest illness I have seen for some time. On May 26 I dictated a note: "Nails and lips very blue indeed, and in the nails is to be perceived without pressure a most distinct capillary pulse." Then on June 10 a note, written by myself, runs: "During the last six or seven days the patient has seemed at times to be dying with circulatory failure; pulse slow, only 66. Oxygen continuously inhaled; has seemed to be of some service in removing the extreme pallid cyanosis. She has frequently been deeply unconscious, and while in this state the lips twitch in a most marked manner, chiefly drawn to the right. While conscious she has complained of stiffness, especially in the right arm and face, and of inability to open the mouth. There is no evidence of thrombosis in accessible situations, but I can frame no other theory than thrombosis of some intra-cranial or deep veins to account for her appearance. The respiratory rhythm has been most markedly interfered with, varying from almost

imperceptible slow movements corresponding to the periods of unconsciousness to very rapid shallow breathing (60 per minute) during consciousness—broadly, a sort of Cheyne-Stokes with hours instead of minutes or seconds to represent the various phases; at times, too, an ordinary Cheyne-Stokes." After this note I considered her moribund and too ill to examine physically, but to my astonishment she gradually improved, the unconscious intervals becoming shorter and with longer intervals between them, the respiration gradually getting back to normal, and only the headache remaining. For a week or two at this time of recovery she had several "fits"—quite unconscious, pupils dilated, but equal and react to strong light. My next written note is dated August 22, and runs—"For last two days patient has complained of very severe pain in the front of both tibiæ and in the calves; the periosteum is distinctly thickened to the touch, but otherwise there is nothing to be seen or felt—no general œdema, no purpura." Heat was no use in relieving this pain; cold in the shape of an ice-bag was more helpful, but as the pain was causing very great suffering and preventing sleep, I ordered incisions to be made through the periosteum to the bone. This gave marked and immediate relief, and the pain has not returned up to date.

From the end of August till now the patient has remained in hospital: her health varying considerably, at one time well enough to sit in the sun, at another so prostrate as to be compelled to keep her bed. On one or two occasions she has had a recurrence of the fits above mentioned, and with them has reappeared the intense cyanosis of the lips and nails; but each recurrence has been speedily followed by what may be termed recovery, though the underlying condition of anæmia shows little, if any, improvement.

Comments.—The case presents three features of interest—

1. Its obstinacy and rebelliousness to treatment.
2. Nervous changes of a peripheral type.
3. Nervous (? vascular) changes of a central type.

1 is perhaps not a very uncommon feature, though I have not hitherto met with such a very extreme case; I could never flatter myself that the treatment had done more than mitigate in a very slight degree the condition of the blood. The menstrual history is closely in accord with Trousseau's old clinical observation as to the relation of early and profuse menstruation with obstinacy of anæmia.

2, again, is by no means an uncommon feature in anæmic

girls; the feature was very marked as regards the trophic changes in the hands—warts and ulceration, and I suppose her present pain in the leg may be considered a form of angioneurotic œdema, though it is somewhat unusual in its situation. Considering the enormous amount of iron she was taking in 1897 and 1898 I wondered if this could be producing a neuritis.

3. It is round this point that the principal interest of the case centres. What was the nature of the vascular disturbances underlying the phenomena? At the time, considering her moribund, I had no hesitation in believing it to be thrombotic, and the œdema of forehead—slight but marked—and the generalised nature of the nervous disturbance (unconsciousness, profound respiratory changes, mouth twitchings, etc.) made me think we had a wide-spread cortical venous stasis, perhaps due to sinus thrombosis—my house physician originally thought this out. The course of her illness, and especially her recovery, compels me, however, to drop this view, as I have had no experience of sinus thrombosis followed by recovery. Throughout the whole four or five years of ill-health, headache has always been a prominent feature, and I feel now compelled to believe that nutritional disturbance of cortical neurons must have been the essence of the morbid changes underlying her unconsciousness and its attendant phenomena, more severe in degree but probably of the same nature as those which are causing the headaches.

Note.—Subsequently to the patient being shown at the Clinical Society, and in consequence of the view then suggested that syphilis might be the cause of the trouble, vigorous antisyphilitic (inunction of Ung. Hydrarg. and Pot. Iod. in rapidly increasing doses) measures were adopted, but unfortunately without any trace of benefit.

The patient again came to me in March, 1900, still complaining of intense headaches which prevent sleep, otherwise she seems in the same condition as for the last four years. The tibial pain has not returned up to date. I intend to have the nasal cavities and frontal sinuses examined.

III.—*A case of Acute Streptococcal Meningitis supervening in the course of Chronic Parenchymatous Nephritis.* By H. D. ROLLESTON, M.D. *Read October* 13, 1899.

A MAN, æt. 22, was admitted to St. George's Hospital on May 26, 1899, with a temperature of 103°, a dry tongue, and a history of eight days' malaise. This history naturally at first sight suggested typhoid fever. The illness began with sore throat, but he had kept at his work—that of a footman—until his admission.

There was entire absence of headache, and the patient's replies to questions were so much to the point that the tentative diagnosis of typhoid fever seemed doubtful. It was found later that Widal's reaction was entirely absent. His pupils were normal, there was no discharge from the ears, he had no sickness, and the heart and lungs were normal. The spleen could not be felt, and there were no abdominal distension or rose spots. The pulse was 112. The urine was fairly copious, of sp. gr. 1016, contained much albumen, casts, a few red blood-cells, and leucocytes.

On the following day he had two epileptic fits; on the next morning he complained of pain in the neck, and later in the day he had two more epileptic fits, and passed into a drowsy condition with evident signs of cerebral irritation. He was restless at times, and tried to get out of bed; there was twitching of the face and of the angles of the mouth. His skin was moist, and the pulse rapid, reaching 130. Cheyne-Stokes respiration developed some hours before his death, which occurred suddenly next morning.

The *post-mortem* examination showed that the intestines were normal, except for enlargement of the solitary follicles and pigmentation of Peyer's patches, and that the chief lesions were (*a*) acute suppurative meningitis and (*b*) an acute attack on chronic parenchymatous nephritis. There was universal suppurative meningitis, evidently of several days' standing; the pus was examined by Dr. Slater, and found to contain streptococci. The ears and the sinuses were quite healthy. The two kidneys weighed 14 oz. and 13½ oz. respectively; the capsules peeled off easily, leaving a smooth

mottled surface. On section the pyramids were markedly congested, while the intervening portions were of an opaque, dirty white colour, due to the tubes being stuffed with white débris. Microscopically there was much thickening of Bowman's capsule; the tubes were dilated, and contained much fatty epithelium; there were conical areas of fibrosis in the cortex, resembling those seen in the kidneys of arterio-sclerosis. So far these changes are those of a contracting white kidney, but in addition there was evidence of acute change, namely, degeneration of the epithelial cells of the convoluted tubes, blood in some of the tubes, and some areas of small cell infiltration. As no streptococci could be found in microscopic sections of the kidneys, the acute nephritis may be regarded as due to changes set up by toxins absorbed from the meninges. But inasmuch as cultures were not made from the spleen and heart's blood, it cannot be certainly concluded that the meninges were the only site of streptococcal invasion.

The heart weighed 16 oz., and showed hypertrophy of the left ventricle. The myocardium was swollen and somewhat pale. The aorta showed a considerable amount of discrete and early atheroma. The lungs were œdematous, but otherwise healthy. The liver weighed 90 oz. and showed cloudy swelling. The spleen, 16 oz., was large and much softened.

Remarks.—The case was one of chronic parenchymatous nephritis in which an acute streptococcal infection of the cerebral meninges had taken place. It is possible that the orifice of inlet for the streptococci was through the tonsils, inasmuch as the patient's illness was said to have begun with a sore throat.

The occurrence of terminal infections in the course of renal disease is well known, and Flexner,* in a very interesting research into the terminal infections that occurred in 255 cases of chronic heart or kidney disease or both combined, examined bacteriologically after death, found that terminal infections had occurred in no less than 213. His observations further tended to show that this disposition to infection depends on the fact that the blood of patients suffering from chronic disease has in great measure lost the bactericidal power present in the blood of healthy persons. While these terminal infections are generally due to the usual pyogenic cocci, it appears from Flexner's figures that in the few cases of meningitis due to secondary infection in the course of chronic renal

* Flexner, "A Statistical and Experimental Study of Terminal Infections," *The Journal of Experimental Medicine*, vol. i, No. 3, 1896.

disease, the responsible micro-organism is almost always *Diplococcus pneumoniæ,* the original inlet for the microbe being pneumonia.

In this case it is remarkable that the cerebral meninges were specially affected by the streptococci. There was no very manifest disposing cause for this, such as local disease, injury, or sunstroke, but possibly the resistance of the meninges was congenitally deficient. This hypothesis receives some support from the fact that a brother of the patient was said to have died from meningitis.

While secondary infection in the course of chronic renal disease often gives rise to acute pericarditis, pleurisy, peritonitis, and endocarditis, it is so rarely that meningitis is thus brought about that this case is worthy of being recorded.

IV.—*A case of Acute Intestinal Obstruction due to an Intussusception of Meckel's diverticulum.* By H. Betham Robinson, M.S. *Read November* 10, 1899.

A HEARTY well-nourished little boy, æt. 5, was seen by me in consultation with Dr. How on January 22. He had apparently always had good health, but on questioning there was a history of occasional complaint of pain in the stomach, unaccompanied by any other symptoms. He had had rather a severe attack of whooping-cough in the summer, but he was now quite free of it. About a fortnight before there had been a "bilious attack," which only lasted a day, and then he was well.

After his breakfast on the 21st he complained of feeling unwell and sick, but his bowels were relieved. Some castor oil was given, after which he vomited. During the day he complained of pain in the stomach, and his temperature was not above normal; in fact there was nothing to point definitely to any serious lesion. During the night he vomited constantly, but only "bilious" vomit, and had attacks of "cramps," the pain being referred to the umbilicus and across the abdomen above this point. He had some hiccough and great thirst, but no flatus was passed.

On the morning of the 22nd he was in much the same condition, if anything a little better, but vomited clear bilious fluid at intervals, and especially anything given immediately afterwards. His belly was not distended, and there was no lump to be felt; nothing was to be felt in the rectum. His temperature was 99° and pulse 130. In the afternoon he was much the same, only the attacks of pain were almost continuous, and the belly was a little swollen, but no tumour could be felt. An enema was given, but nothing was passed; it was noticed, however, that the returned fluid showed a little blood-staining. He had extreme thirst and vomited everything, but the vomit was still only bilious.

I was fetched to see him in consultation, and found the following condition at about 9 o'clock. He had a distressed look and was extremely restless, lying in bed with his knees drawn up. He was constantly crying out with pain, which he referred about the umbilicus and across the belly. His tongue

was moist. The temperature was 99°, and the pulse 160 and compressible. He vomited directly anything was given to him, but the vomit was clear, watery, and bile-stained. No flatus was said to have been passed, and the urine had been voided in small quantities.

On examining his belly it was distended, the coils of small intestine being very evident in the upper part above the navel; there was no special prominence anywhere. On palpation there was no rigidity anywhere, and apparently not much tenderness; no tumour could be felt except an ill-defined lumpiness just above the outer part of Poupart's ligament on the right side. The note over this was comparatively dull, this diminished note passing into the right lumbar region, but elsewhere the belly was resonant. Nothing was to be felt *per rectum*, but the fæcal smear on the finger was noticed a little blood-stained: there was no mucus in the bowel.

It was very evident that we had here a case of obstruction requiring immediate operation, the nature of which was indefinite; the symptoms seemed like intussusception, but there was no lump, and absence of the classical sign of blood-stained mucous discharge.

Operation was proceeded with at 10.15, the child having chloroform administered. On examining the belly then there was no rigidity and no tumour to be made out, but *per rectum* a lump was felt in Douglas's pouch, which could be pushed up by the finger and then felt with the other hand above the inner part of Poupart's ligament to the right of the bladder. It seemed firm and tubular or sausage-shaped, its length as gauged between the fingers being about two and a half inches. Its low situation and free mobility were rather puzzling. There was nothing in the cæcal region.

The incision was made in the middle line about two and a half inches long, and on opening the peritoneum some clear fluid escaped but no lymph. A coil of dilated small intestine presented which was pushed back, and two fingers were passed down into Douglas's pouch, when they gripped the lump and drew it out of the wound. With sponges the intestines were prevented escaping. It could then be seen that it was intussuscepted small intestine, in direction from left to right, forming a tumour fully four inches long, concave, with an enlarged gland on the left face of its mesentery. Without much difficulty the main part of the gut was reduced until a hard lumpy mass was displayed with a cup-shaped depression in its centre; a little more reduction showed that the normal

axis of the intestine was restored, and that the lump came from its free margin. This could be nothing else but an inverted Meckel's diverticulum. Reduction so far had been accomplished without any splitting of the serous coat, but any attempt to reduce the still inverted diverticulum, which remained as a considerable lump in the bowel, caused rents in the serous layer about its thickened and somewhat contracted base. There was noticed passing off the base of the diverticulum over the bowel to the right layer of the mesentery a ridge of peritoneum, evidently its mesentery, and on the left side was a similar ridge, only smaller.

The bowel wall at its junction with the diverticulum on its left side looked doubtful. No adhesions were met with over the unravelled gut, and its serous layer generally had a good polish.

The only treatment possible was to excise the diverticulum, which was done by cutting round its base. The edges of the wound in the bowel thus produced were brought together by Lembert's sutures. Owing to the doubtful state of the bowel at one point, it was thought better not to entirely close it but to fix it in the wound. The boy's condition forbade any extensive resection, and, moreover, it was unnecessary, as only part of the lumen of the bowel had to be sutured together. The operation lasted a little under an hour. With hot bottles and brandy by enema and subcutaneous injection he revived considerably, but after nearly two hours he began to fail suddenly and died.

The specimen on examination showed that the diverticulum was about three inches long, ending in a blunt extremity, and that its lumen was very little smaller than that of the intestine. The end of the inverted diverticulum was capped by a false membrane, and the walls generally were considerably thickened, but showed no signs of destruction.

V.—*On a case in which Primary Nephrectomy was performed for Complete Rupture of a Kidney.* By J. Bland-Sutton. *Read November* 10, 1899.

ON the evening of July 17, 1899, a man æt. 35 tried to stop a runaway cab horse. He was knocked down and the vehicle (a four-wheeler) passed over him.

On admission into the Middlesex Hospital the man was semi-conscious, very pale, and collapsed. The respirations were quiet and of normal rate; his pupils were dilated and the extremities cold, and he complained of great pain in the right loin.

I saw him three hours after admission. The pulse was 96 per minute and fairly full. The temperature 96° F., and he had not rallied since admission. The man complained of great pain in the right loin, which seemed to be occupied by a tense, ill-defined swelling. His condition indicated severe bleeding from an abdominal viscus, most probably the right kidney. There were no scratches or contusions on the skin of the belly.

As the patient's condition seemed very grave I decided to arrest the bleeding if possible, and whilst arrangements were made for the operation a catheter was passed and three ounces of blood-stained urine drawn off.

By means of an incision in the right linea semilunaris I satisfied myself that the liver and intestines were uninjured; then by detaching the peritoneum from the outer margin of the incision I easily exposed the swelling in the renal region, turned out a large mass of clot and bright arterial blood. On exposing the kidney its lower one third was found completely detached transversely from the upper two thirds and merely hanging by the renal vein and ureter. When the parts were drawn up blood issued in a large volume and with some force from the renal vein, which had been torn in the direction of its length. I examined the parts carefully with the hope that the two portions of the kidney might be sutured and the organ saved, but the extensive laceration of the vein contra-indicated such an attempt at conservative surgery, so I transfixed the pedicle in the usual manner and removed the kidney. After carefully removing the clot remaining in the peri-renal

region, it was lightly stuffed with gauze and the wound secured with interrupted sutures of silkworm gut and dressed with sterilised gauze. The patient bore the operation well, and it was not necessary to resort to transfusion. The subsequent course of the case did not afford anything worthy of comment and ended in excellent recovery, the patient being sent to a convalescent home within four weeks of the operation, and subsequently reported himself with a very narrow sinus.

Remarks.—The facts of this case are very simple, and the conditions found at the operation were such that one may venture to believe that there could be no difference of opinion in the minds of surgeons in regard to the mode of treatment adopted. It may be that this accounts for the extreme paucity of records in regard to this injury in British surgical literature. I cautiously framed the title of this communication "Primary Nephrectomy for Injury to the Kidney," because it should be used to cover cases where this operation is carried out within twenty-four hours of the accident. I know that nephrectomy has been necessary several days, and even many weeks, after an accident to the kidney; in such circumstances the operation should be termed "secondary nephrectomy," and as being in accord with the usual method of adjusting terms in surgery.

Our "home" literature contains remarkably few recorded cases of primary or secondary nephrectomy for injury. A few cases find their way annually into the excellent and very useful *Centralblatt für Chirurgie*. From this source I have gleaned some information which may be briefly expressed thus:

Rupture of the kidney, whether it be due to the passage of wheeled vehicles over the abdomen, falls from heights on to narrow bars, rails, or narrow hard edges of any kind, or kicks from horses, generally excites astonishment in those who deal with the case that such extensive and often mortal injury to a protected organ like the kidney can be effected with such slight, and often entire absence of injury or abrasion, of the overlying skin. This is a clinical fact to bear in mind.

The accident seems to be more common in children than in adults; perhaps a larger series of cases than is at present available would not confirm this. It is one that would repay a careful search through current surgical literature.

It is also necessary to emphasise the fact that many of the

records are from the post-mortem room, the patient dying without surgical aid; this is regrettable.

Lastly, a perusal of the few available records proves that an individual may receive a severe and even mortal injury to the kidney, death being the direct result of hæmorrhage, without blood appearing in the urine, though the patient may survive the injury long enough to pass urine in fair quantity. Though such a condition of things is, I believe, uncommon, its occurrence rests on good testimony.

In the case described in this communication the clinical facts were very definite, namely, the passage of a wheeled vehicle across the abdomen, followed by signs of severe internal bleeding, and the formation of an ill-defined swelling in the loin absolutely dull on percussion, and in the course of a few hours the presence of blood in the urine. Severe bleeding from an abdominal viscus due to injury invariably repays prompt surgical intervention, and the number of cases which die from bleeding consequent on rupture of the kidney seems to me larger than it ought to be.

Note.—In December, 1899, the sinus had closed.

VI.—*A case of Enlarged Spleen, due to Congenital Volvulus of the Stomach and Transverse Colon, and simulating Splenic Anæmia.* By NORMAN DALTON, M.D. *Read November* 10, 1899.

H. B., a woman æt. 38, was admitted into King's College Hospital on December 16, 1898, for anæmia with enlargement of the spleen. She was married, and had had three children. Her family history was good. She had had rheumatic fever when sixteen, and some chronic rheumatism when twenty. In other respects her health had been good, and there was no history of malaria, syphilis, or alcohol.

She had noticed that her abdomen remained large after her first child was born, eight years ago, but no tumour was discovered until December 11, 1898, when she consulted Dr. Yencken of Peckham, who found her suffering from jaundice, with a temperature of 102·6°. On examination he detected the enlarged spleen.

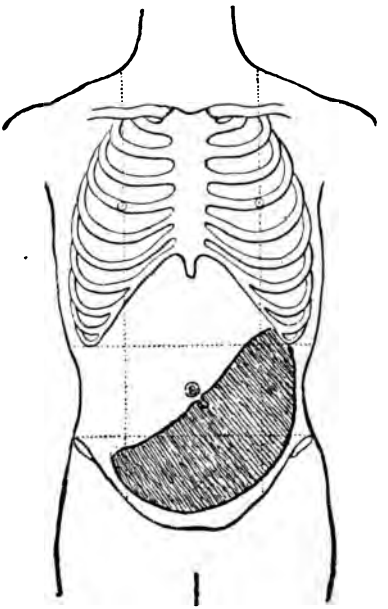
When admitted the patient said that she had been getting weak and thin for a year, and had noticed an increasing pallor for six months. She thought that the abdomen had not increased in size during this time, and she had had no pain in that region whatever.

The jaundice was now scarcely noticeable. She was very pale, and neither thin nor fat. There was a venous hum in the neck, and a systolic murmur over the pulmonary artery and at the apex of the heart. The lungs were normal. The spleen was very large. Its upper end was rather lower than normal, being situated just behind the lower margin of the ribs. From this position the organ could be felt quite distinctly, occupying the left lumbar and iliac regions, the hypogastrium and the lower umbilical region, and the right iliac fossa. One of the notches in its anterior edge could be felt at the umbilicus, and the whole of the organ was hard, perfectly smooth, and not tender, except, perhaps, at its posterior part in the left lumbar region. I may say here that it did not alter appreciably during the six weeks the patient was in the hospital. The liver was not felt, and there was no ascites. The urine was normal except that there was always a heavy deposit of urates, probably due to the fever. One or two

glands in the neck were slightly large and hard, but in no way suggestive of lymphadenoma. The eyes and the pelvic organs were normal.

The blood showed marked oligocythæmia, the red blood-corpuscles numbering 1,060,000 per cubic millimetre (21 per cent.). The hæmoglobin was reduced to 20 per cent. There was no leucocythæmia,—in fact, although the proportion of white to red discs was as 1 to 220, the total number of leucocytes

FIG. 2.



To show the size and position of the spleen.

was about half what it should be, namely 4800 per cubic millimetre. There were no abnormal corpuscles, except a few coarsely granular eosinophils such as might occur in any cachectic state.

While in the hospital the temperature varied from 102° to 99°. Her condition did not alter appreciably, but she gained seven pounds in weight. She was treated with arsenic, iron, and quinine. At times she complained of flatulence and of pain in the back. Menstruation occurred once.

On the morning of January 22, 1899, she suddenly became collapsed, with pain in the abdomen. The temperature fell to 97°, and the pulse became almost imperceptible. Without going into details, I may say that her symptoms pointed both to perforation and to hæmorrhage. The only special point was a very great distension of the stomach, which pushed the spleen a little to the left, but the spleen itself was no smaller. She died in a few hours.

At the *post-mortem* the thoracic organs and the liver and the kidneys were normal. The peritoneal cavity contained gas, shreds of food, and a great deal of highly sanguineous serum. There was very little peritonitis.

In describing the relations of the abdominal viscera I shall, of course, assume that the body is in the erect posture, as is done in all anatomical descriptions.

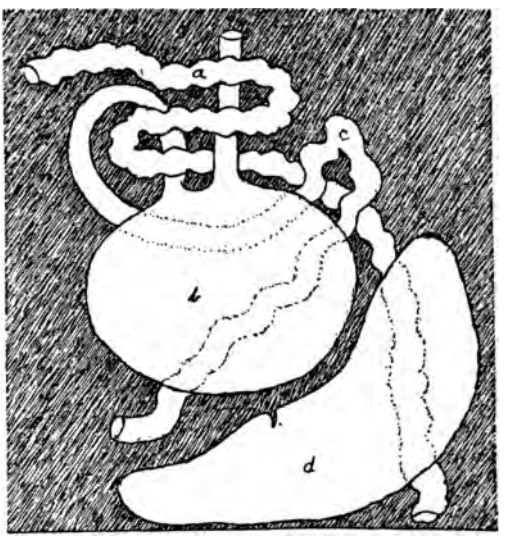
On opening the abdomen nothing could be seen but the spleen and the stomach. The former occupied the position which was marked out during life, and the latter filled the rest of the abdominal cavity, as then seen. On further inspection the stomach was found to be ecchymosed, so that it resembled a strangulated piece of intestine. It was also extremely ballooned, and rotated in such a way that the greater curvature was directed forwards, and formed a line which sloped downwards from left to right.

On pulling aside the stomach it was seen that the transverse colon lay across the epigastrium on a higher level than the lesser curvature of the stomach. It had no mesentery, but was closely attached to the posterior abdominal wall, in the same way as the ascending and descending colon are attached. The next thing observed was that the omentum which hung from the stomach was not reflected in the normal manner, but ended in a free margin; and that the piece of omentum which hung from the transverse colon also ended in a free margin. Now this is the condition in the fœtus until about the fourth month, at which time the omentum attached to the stomach unites with that attached to the transverse colon to enclose the cavity of the great omentum of adult life. Consequently the reversal of the normal position of the stomach and transverse colon was congenital.

As regards the stomach, there was about an inch of œsophagus dragged down through the opening in the diaphragm into the abdomen. This piece of œsophagus was crossed from right to left by the transverse colon, which lay in front of it. In the left hypochondrium the colon was acutely flexed,

and, turning back again, it crossed in front of the œsophagus a second time, but now, of course, from left to right, as will be seen in the diagram. The colon then bent sharply behind the cardiac end of the stomach, and passed first to the left and then downwards, becoming the descending colon in the usual position behind the spleen. In the diagram I have been obliged to draw the bendings of the colon as if the coils did not touch, in order to make the relations clear, but it must be understood that all the coils were closely in contact, and

FIG. 3.



a, the transverse colon. *b*, the stomach. *c*, coils of small intestine. *d*, the spleen.

that the winding of the colon round the junction of the œsophagus and stomach was very tight. At the post-mortem, any traction on the piece of colon to the left of the stomach, and any traction on the spleen (especially on its lower part), strangulated the cardiac orifice of the stomach completely.

The pylorus was found to be close to the cardia and partially behind it, and the stomach was rotated, just as if, standing in front of the body, you had taken the fundus in your right hand and the pyloric end of the greater curvature

in your left, and twisted the viscus round with the fundus in front; in fact, the cardia and pylorus were half twisted round each other. Just at the left side of the cardia, where the constriction was very tight, there was a perforation of the stomach, clearly produced by sloughing.

As regards the spleen, the gastro-splenic fold of the peritoneum was behind the stomach, and was obviously stretched and twisted. The spleen itself was of an uniform deep red colour, and firm, without being extremely fibrous. The Malpighian bodies were not visible to the naked eye,—in fact, no white spots of any kind were to be seen. In the portal vein there was a quite recent thrombus.

In the left hypochondrium there were several coils of small intestines, which had reached that region from beneath the stomach, and had crossed in front of the beginning of the descending colon. They lay between the diaphragm and the top of the spleen, and partially covered the bendings of the transverse colon.

Such being the relation of the parts, I believe that the sequence of events was as follows:—The twisting of the colon round the cardiac end of the stomach was congenital, but caused no inconvenience at first. During the first pregnancy the spleen became loose, and here I may remind you that, as Bland-Sutton has pointed out, most cases of wandering spleen date from pregnancy. Once loose, the spleen would drag on its ligaments and narrow its own vein, and thus become larger and larger from congestion. Its weight would also drag the stomach downwards. When the spleen was large enough to reach the right side of the abdomen, its most pendulous part, which then lay in the right iliac fossa, would by its ligamentous attachment to the left border of the cardia drag that part of the stomach against the encircling colon. At the post-mortem this could be clearly demonstrated by exerting traction on the lower part of the gastro-splenic ligament. This traction constituted a permanent risk of strangulation, but, partly perhaps because the spleen was supported by the right iliac fossa, the food was able to enter the stomach.

There were thus two sources of trouble, first the winding of the colon round the cardia, and second the weight of the spleen, which tended to make that winding tight. But a third trouble existed, namely the proximity of the cardia to the pylorus, for, whenever the stomach became distended, the rotation of the fundus to the front and right would tend to obstruct both cardia and pylorus by producing a twist.

As long as digestion was normal no serious consequences resulted from the three abnormalities mentioned above. When the stomach was empty the cardia and pylorus were untwisted, and the food could enter the former orifice. A moderate amount of distension and consequent twisting would then occur, but not sufficient to prevent the exit of food through the pylorus, though probably some feeling of indigestion was always present. But eventually the gastric digestion became impaired, and the stomach became greatly distended by gas after each meal. This would cause the rotation of the stomach and the intertwisting of the cardia and pylorus to become extreme, and as both orifices were closed, the gas could neither be eructated nor passed into the duodenum. This readily explains her constant complaint of flatulent distension, and it also explains the absence of vomiting. Eventually some exacerbation of the twisting, aided by some tightening of the colon round the cardia, produced an absolute strangulation, which ended in sloughing of the stomach wall.

The faulty condition of the stomach might account for the anæmia, because, during the last few years of the patient's life, the food would enter the duodenum with great difficulty, and after it had been exposed for a long time to fermentative processes in the stomach. The fever is less satisfactorily accounted for, and I can only suppose that it was caused by the irritable condition of the stomach, and the absorption from that organ of toxins produced by bacteria. It is probable that the spleen became smaller during the later pregnancies, because the uterus would push it up and support it from below, and so relieve the narrowing of the splenic vein. After delivery it would, of course, soon regain its former size.

I did not recommend an operation when the perforation occurred, because I thought that the patient would die if moved, and I do not think that it would have been possible to disentangle the parts during life. But I much regret that I did not recommend splenectomy soon after she entered the hospital, because the removal of the weight of the spleen would have relieved the twisted tubes to a great extent; and she would have probably lived in comfort for years if no great cicatricial contraction had occurred after the operation.

As regards diagnosis, during life the case was only considered from the point of view of the spleen and the anæmia. It was easy to eliminate leucocythæmia, lymphadenoma, malaria, syphilis, and lardaceous disease. Tumours and tuberculosis of the spleen and infarctions could be excluded

by the duration of the case, the absence of pain and tenderness, and the perfect regularity of the surface of the viscus. The last point also excluded hydatid cysts. Obstruction of the portal vein itself was excluded by the absence of ascites and of other signs of portal obstruction, and, in addition, such a very large spleen is seldom caused by portal obstruction. So that by a process of exclusion there remained only two possible diagnoses. The first was that the enlargement of the spleen was due to obstruction of the splenic vein itself, and the second was that the case was one of so-called splenomegaly.

I may say that I inclined to the former view, but this did not constitute a diagnosis, because I was unable to think of any cause of obstruction of the splenic vein which would also cause the anæmia and the fever. The fact that the enlargement was noticed after pregnancy suggested that the spleen was an ordinary wandering spleen, with a twisted gastro-splenic ligament; but, although a certain amount of anæmia might arise from this condition, it is hardly likely to be so severe as in this case, and further, no rise of temperature should have occurred. The same objections applied to the theory of thrombosis of the splenic vein. A malignant growth in the stomach might have compressed the vein, and might have caused the anæmia. But it probably would not have caused the fever; and, in addition, the absence of any tumour of the stomach was strongly against this view; in fact, there were no signs of tumour, aneurysm, or abscess anywhere in the neighbourhood of the splenic vein.

A somewhat plausible theory which occurred to me was that a chronic ulcer had perforated the posterior wall of the stomach, and caused a fistula in the connective tissues behind it. The suppuration of the fistula would, on this supposition, have accounted for the fever, the ulcer would have partly accounted for the anæmia, and the cicatricial tissue around the ulcer might easily have compressed the splenic vein, or have caused thrombosis within it. Naturally, however, I did not have much confidence in so complicated a theory, and the case remained a mystery until the sudden and unexpected death of the patient occurred. The post-mortem then revealed the real state of affairs, which I think I may claim to have been quite undiagnosable during life.

It will have been noted that the symptoms presented by my patient closely resembled those of the disease called splenic anæmia or splenomegaly. The great enlargement of the

spleen, the remittent fever, and the extreme anæmia with oligocythæmia and chlorosis, but without leucocythæmia, constitute the characteristic features of that affection. Against the diagnosis of splenomegaly was the fact that the hæmoglobin although more diminished than the red corpuscles was not very much so. Also the liver was not enlarged and no hæmorrhages had occurred. Still, I would have been obliged to put down this case as one of splenic anæmia, if there had been no post-mortem. In this respect it would have been on a par with Dr. Sidney Coupland's case, which was thought to be one of splenic anæmia until the post-mortem proved it to be one of syphilitic liver. I will add that I am, at present, by no means convinced that the condition called splenic anæmia is a real pathological entity, as Banti considers it to be. My reasons are that the total number of cases on record is still small, and that in several of these, including some which were most accurately observed during life, no post-mortem was obtained. Further, some of Bruhl's cases are quite unconvincing; and finally, in one or two cases the post-mortem has shown that the symptoms could be explained without calling into existence a new disease.

VII.—*A case of Senile Tuberculosis.* By Howard Marsh. *Read November* 24, 1899.

THE following is so remarkable an illustration of tuberculosis in the old—the senile scrofula of Sir James Paget—that I venture to offer a brief record of it, especially as the subject, which is one of much interest and importance alike from the standpoints of diagnosis and treatment, has never before, I think, been under discussion at any meeting of this Society.

In June, 1894, Miss R., who was then 71, and a patient of Dr. Neale of Hampstead, noticed swelling and pain in her left ankle. These symptoms continued, and soon the integuments became dusky red and over-warm, and the surrounding parts œdematous. Suppuration occurred in the following October, and the joint was opened and drained. Discharge, however, continued, and it was found that pus was burrowing up the leg in the sheaths of the flexor tendons; muscular wasting took place, pain became severe, and the patient's health and strength markedly declined. Amputation through the lowermost third of the leg was therefore performed in March, 1895. The wound shortly healed, and the patient recovered her general health.

At the end of twelve months, during which time the patient had been in good health, periostitis was developed in the lower half of the left ulna, attended with pain, considerable swelling, and dusky redness and œdema of the integuments; and in the course of three weeks suppuration was detected. The abscess was opened and half an ounce of pus let out, and the cavity cleared of granulation tissue. Very slow repair followed, and a small sinus remained for three months. It then slowly healed. Nine months after this date —the patient being then seventy-three—the right ankle became swollen and painful, and in spite of all that could be done the joint, passing through identically the same changes as had been noticed in the opposite joint two years before, went on to suppuration. Pus was evacuated by aseptic incision, and free drainage established. But all to no purpose; for matter burrowed upwards into the calf, the integuments became dusky and œdematous, and the general health

declined. Amputation was performed in March, 1897, and within five weeks the wound was soundly healed. In June, 1897, an abscess formed over the ulna in the site of the periostitis in 1896. This was opened, and it slowly healed.

Eighteen months now elapsed, during which period, although increasing age told somewhat on her strength, the old lady remained quite well. Then in August, 1898—she being now seventy-five—her right knee swelled, and became so painful that she could sleep very little during the night, while during the day she was in constant suffering. At first she was relieved by a carefully-adjusted back splint, which Dr. Neale applied. It soon, however, became evident that the disease was steadily and, indeed, rapidly progressing, swelling increased, the joint was hot, the surface was dusky, and the soft parts œdematous, and painful startings followed even the slightest disturbance of the limb, or any attempt on the part of the patient to go to sleep. She looked ill; the temperature was 101° to 102° at night; the pulse was feeble and becoming rapid, and she lost flesh. The only chance of saving the patient's life lay in amputation through the lower third of the thigh; and this operation was done in December, 1898, four months after the knee was attacked. The operation was well borne, and the wound healed without suppuration in the course of six weeks. Since this date the patient has been very well, and has had no further development of tuberculous disease.

The three joints which in the course of this case had been removed by amputation were found, on dissection, to have undergone changes which were practically identical in all. The disease had in each instance evidently begun in the synovial membrane, and had proceeded to the complete destruction of the ligaments and of the greater part of the articular cartilage, so that only shreds and thin flakes were here and there to be seen. In some situations the ends of the bones were ulcerated. The cavity of the joint was occupied with an accumulation of tawny-coloured and friable granulation tissue and cheesy pus, while the soft structures around and the neighbouring tendon-sheaths were infiltrated with pus, which, in the absence of firm granulation tissue, was free to extend in every direction.

In all I have seen of senile tuberculosis—and the older the patient the more evident has it been—this tendency towards progressive and, usually, rapid destruction of the joint involved has been a marked characteristic of the disease.

In the old the capacity of the tissues to resist the tuberculous process seems diminished almost to the vanishing point. They readily break down and become the seat of suppuration, in the course of which large quantities of pus are rapidly formed, entirely apart from septic infection. Diagnosis, in many instances, is at first difficult, for the symptoms may closely resemble those of osteo-arthritis—pain, stiffness, moderate swelling, muscular wasting, and sometimes creaking or grating on movement. But before long, often in the course of two or three months, or even from the first, the joint is over-warm, the surrounding soft structures are infiltrated, pitting on pressure is detected, and, when the joints are superficial—the elbow, wrist, knee, and ankle—there is a suffused and dusky appearance of the skin.

In some cases, however, these symptoms are absent; and then the true nature of the case only becomes apparent when suppuration takes place. As to suppuration, my experience is that it occurs in a large proportion of cases of senile tuberculosis, and that it tends to be persistent and copious, and to produce extensive and rapid destruction of all the structures of the joint. Prognosis is very unfavourable. Once it is involved in this disease a large joint, so far as I have seen, is almost certain to be destroyed; indeed, I cannot recall an instance in which repair took place. Amputation is usually the only course to follow.

VIII.—*A case of Excision of the Cæcum for Carcinoma of the Ileo-cæcal Valve and Obstruction.* By Arthur E. Barker. *Read November* 24, 1899.

W. B., æt. 30, a plumber by trade, was admitted into University College Hospital, under the care of Dr. Risien Russell, on May 5, 1899, suffering from colicky pains in the abdomen and limbs; these had been present on and off for three months, but more severely for the last fortnight. He had also suffered from constipation for three months with loss of appetite and occasional vomiting. He had no symptoms of lead poisoning and no appreciable wasting. Latterly the bowels had entirely ceased to act for considerable periods, and on the present occasion the concomitant symptoms of obstruction had become so marked that Dr. Russell felt that the time had come for surgical interference. He was, therefore, transferred to my ward on May 7 for operation. When I saw him his general condition was good. The pulse and respirations were regular, and he complained of but little more than distension and vomiting, but neither of these symptoms was extreme. The abdomen was fairly tense all over, but without particular tenderness at any spot, and there was no definite tumour to be made out either through the rectum or abdominal wall. The pulse ranged from 88 to 104, the temperature between 99° and 100°. He had vomited five times on the day before operation, and after an enema a certain amount of fæces had come away, but without relieving the distension. There appeared, then, to be some cause of chronic obstruction with more acute exacerbations grafted on to it. Under the circumstances no definite diagnosis could be made, and it was determined to explore the abdominal cavity.

Operation May 7, 1899, close upon midnight. A median incision was first made (under local anæsthesia, followed later by chloroform) below the umbilicus. The small intestine was at once seen in its lower coils to be enormously distended, being at least four inches in diameter; it was also greatly hypertrophied and slightly injected. As it was impossible to further explore the abdomen in the presence of this distension, I at once opened the bowel and evacuated about five pints of

fœtid liquid fæces. A clamp was now placed upon the opening and further exploration was possible, but first it was necessary, in order to explore a hard lump which could be felt in the ileo-cæcal region, to divide the right rectus transversely outwards two inches below the umbilicus. I was then able to see that the ileo-cæcal valve was surrounded by a mass, above which the greatly hypertrophied and distended ileum could be seen to terminate; below it the cæcum and colon were empty and thin-walled. All this had taken time, and I was unwilling to add to the loss by the addition of the long operation of removing the cæcum, which would have been particularly dangerous in the presence of much fœtid fæces still coming down from the small intestine. I therefore selected a spot in the hepatic flexure of the colon and in the lower part of the ileum about two feet from its termination, and, making a free opening in each longitudinally, united them in the usual way by two rows of fine silk, a superficial and deep. After thorough cleansing and closure of the original drainage-opening in the small bowel, which had been provisionally clamped, I sutured the abdominal incisions by means of buried and superficial sutures without drainage.

The patient's condition during the operation was satisfactory, and there was but little shock subsequently. The rectal temperature rose for the first two days to 101·2° and 101·6°, and then declined to below 100°. He was fed with fluid peptones by the mouth every four hours, and was also given a little brandy with soda water. In addition, rectal feeding and hot water enemata were carried out regularly. Steady improvement was the result, and on the seventh day there were three normal stools, and two on the eighth day. The vertical part of the abdominal wound healed by first intention; the transverse part suppurated superficially. In the pus, so produced, the organisms after cultivation were found to be mostly staphylococci and not *B. coli* as I had expected.

The patient left hospital on June 6, to recruit for the second operation of removal of the cæcum, which he strongly desired. He then looked very well, and weighed 8 stones 7½ lbs. There was still a small superficial wound over the transverse incision.

On June 21, 1899, six and a half weeks after the first operation, the patient returned to hospital for the removal of the cæcum. He now looked particularly well, ate and drank as usual, and passed his motions regularly; his weight had

increased to 9 stones 3 lbs. There was still a slight granulating patch over the transverse cut of the former operation.

Second operation, June 23, 1899. An incision six inches long was made on the right side parallel to Poupart's ligament, commencing well above the anterior spinous process. On opening the abdomen through this, the tumour was easily exposed, surrounded by much fat. The previous anastomosis was then examined, after separation of some omental adhesions, and was found quite satisfactory. The terminal portion of the ileum was now clamped between two pairs of Doyen's forceps, and divided transversely well below the anastomoses. The proximal end after being cleansed was closed in the usual way by two rows of silk sutures, the last row turning the first well inwards. The mesentery of the lower part of the ileum and of the cæcum was then divided step by step, the vessels being secured as the dissection progressed. Finally, the ascending colon was clamped transversely with two pairs of Doyen's forceps and divided between them, and the tumour was lifted out. The end of the colon was then closed by two rows of sutures applied as in the case of the ileum. After careful cleansing and drying the large wound in the right flank, it was stuffed with iodoform gauze left hanging out of the wound in the abdominal wall. The latter was only closed at its ends, being left well open in the middle. A sterile gauze dressing completed the operation.

The portion removed consisted of the last few inches of the ileum, the whole of the cæcum and a little of the ascending colon. The very hard mass surrounding the ileo-cæcal valve almost completely closed the latter. Its inner surface was not ulcerated, but was very irregular, owing to many almost polypoid excrescences which projected into the cæcum. One of these was as large as the last joint of the thumb. On the outer aspect of the mass was much shrunken fat and hard fibrous material with a few enlarged glands embedded in it. A section of one of the rugged masses in the bowel wall gives the characters of carcinoma, but not unmistakably in my opinion; I venture to hope still that the tumour may be of inflammatory origin. Our surgical registrar, Dr. Curtiss, however, who made the sections, and others who have seen them, pronounce them to show the characters of carcinoma.

The patient bore the operation well on the whole, but while the cæcum was being dragged upon the pulse was observed to become feeble and rapid, though it quickly recovered after the excision of the latter. Under the usual

means of stimulation the patient was fairly comfortable in a short time. There was some bilious vomiting during the night, but the next day it diminished. The wound was dressed on the evening of the second day, and a good deal of clear odourless serum flowed out after removal of the stuffing. From this on to the fifteenth day there is little special to record except steady improvement, but on this day foul pus was observed in the angle of the wound still kept open with gauze. This was the precursor of a small fæcal fistula, which after discharging for some weeks gradually dried up, so that when the patient was obliged to leave the hospital during its closure for the month of August, it discharged very little. At this time the patient was looking well and fat, and his bowels were working regularly. When seen at the end of September there was still a moist point visible, out of which the end of a ligature projected. When this had been drawn out, perfect healing was complete in a few days.

The patient is now at work, strong and well, and eats and drinks as usual, the bowels acting regularly.

The case just recorded appears to me to have a two-fold interest; in the first place, because of the rarity of malignant growths of the ileo-cæcal valve, and secondly, on account of the surgical procedure which was necessary for the saving of the patient's life.

In regard to the first point there appears to be a considerable amount of evidence. But very few cases of carcinoma of this structure appear to have been recorded. If we turn to the *Transactions* of the Pathological Society, we will find very few cases of the disease. In 1891 Dr. Hawkins recorded an interesting case of the kind in these *Transactions*, and incidentally refers to all the cases hitherto reported to the Society. These only amount to some three or four, and since then no further cases appear in the *Transactions*. Dr. Hawkins at the same time quotes from Drs. Fenwick and Bristowe to show the rarity of malignant and other obstructions of the termination of the ileum. For my own part, I have only seen three cases of the kind. One was a patient of Sir William Jenner on whom I was called to make a post-mortem examination, another was placed in my hands by Sir Charles Gage

* "Ulceration of Ileo-cæcal Valve," *Path. Soc. Trans.*, vol. xxxiv, p. 112, Norman Moore.

† "Columnar Epithelioma of Ileo-cæcal Valve," *Path. Soc. Trans.*, vol. xlii, p. 105. Mott, *ibid.*, "Stenosis from Ulceration with Polypoid Masses."

‡ Dr. Hawkins, *Path. Soc. Trans.*, vol. xlii, 1891, p. 132, "Carcinoma of the Ileo-cæcal Valve."

Brown for a large iliac abscess which formed round a carcinomatous growth of the valve, and the third is the present case. If we look to foreign sources of information we find the same rarity of carcinoma of the ileo-cæcal valve. It appears, therefore, to be one of the rarest forms of obstruction in this region.

And if we turn to the surgery of the cæcum we find, as might be expected, that the cases of excision of the organ are few and far between. In the *Transactions* of this Society I can only find one such case, viz. that very interesting one reported last year by Mr. Q. Silcock, in which he excised the cæcum for irreducible intussusception with a brilliant success. Looking abroad, I notice several such cases of excision. One operator, Professor Körte of Berlin, showed me last Easter nineteen or twenty cæcums which he had excised for various morbid conditions, with only one death. His series included cases of carcinoma, tubercle, actinomycosis, and others. Czerny, of Heidelberg, has also put cases on record in which he has excised the cæcum.

As to the method adopted in this case, it will be noticed that I was driven to dothe operation in two stages. The patient was suffering when I was called in from more or less acute intestinal obstruction grafted on a more chronic form. And to have undertaken the excision of the cæcum in addition to the anastomosis for the relief of the obstruction, would, I feel almost certain, have been to make too great a demand upon his resources. This of course opens up the whole question as to the desirability of doing such operations *in two stages* as a rule of practice, a point which might be usefully discussed, I think, by this Society.

I do not wish to enter upon the much-disputed point as to the merits of Murphy's button *versus* simple suture in such cases. I will only remark that I have used the button and the simple suture in many cases, both with success and failure, and the impression left upon my mind is that the simple suture, as used in this case, is the best method to pursue.

As to the prognosis in cases so dealt with by excision for carcinoma, it is too soon to speak certainly in the present case. But I know that in one case at least where such an excision was done by Professor Körte for carcinoma, the patient was alive eight years after. In the present case, at all events, not only was the obstruction, which would have been inevitably fatal, relieved, but a growth was removed widely which could hardly have failed to shorten the patient's life if left untouched.

IX.—*A case of Obstruction of the Inferior Vena Cava, probably Syphilitic.* By CHARLES W. CHAPMAN, M.D. *Read November* 24, 1899.

THE patient whose case I am about to relate was shown at a Clinical Meeting of this Society last session, and the case is recorded in the thirty-first volume of the *Transactions*. I thought, however, that it would interest the Society if I entered more fully into the details than was possible when so many cases were waiting to be seen. I also thought that possibly members might be glad of a second opportunity of seeing the patient.

C. K., a hairdresser, æt. 48, was admitted into the National Hospital for Diseases of the Heart, under my care, on October 6, 1897. He complained of occasional palpitation, shortness of breath on exertion, and of a shooting pain in the head. He further complained of a sense of suffocation when he leaned forward.

History.—Twenty years ago he had a small itching sore on his penis, which caused him no inconvenience. He was treated for two months, and he has not to his knowledge had any trouble from it since. His first illness of any note was influenza in 1892, and the attack was a severe one. Six months after he began to suffer from palpitation; three months later with a sudden sense of fulness in the head, accompanied with swelling and redness of the face, which soon passed off. About this time he saw a doctor, who called his attention to some fulness in the superficial veins of the abdomen which he himself had not noticed. During the attacks of palpitation he would sometimes become suddenly faint, fall down, and remain unconscious for a few seconds. The veins gradually enlarged, and later on the superficial veins of the right leg became varicose. There was no history of sudden strain or any injury to the abdomen.

Examination.—The patient was well nourished, with a dark red face. The veins of the right leg, and those on the abdominal wall, were extremely dilated and tortuous; there was one large vein extending up the right axilla; the left leg was affected in a minor degree. The cardiac dulness was not

increased to the left, and the apex-beat was in the fifth space three quarters of an inch inside the nipple line. Over the sternum the dulness extended well to the right of the middle line of that bone, and upwards to the manubrium. There was no epigastric pulsation. There was a systolic murmur, harsh in character, at the sternal margin of the fourth left intercostal space; a soft systolic murmur was heard at the apex, which could be traced to the anterior axillary border on the same side; the second sound in the pulmonic area was accentuated. The impulse was feeble. Lungs normal. Liver a little enlarged. Urine normal. Tongue furred and indented. Pulse 54. Temperature 90°. There was a hard swelling at the back of the neck the size of half a walnut.

A provisional diagnosis of obstruction of the inferior vena cava from pressure of a gumma or to thrombosis from phlebitis was made. Increasing doses of iodide of sodium were prescribed, and blue ointment was ordered to be rubbed in over the lump at the back of the neck. Under this treatment the symptoms were relieved, and the general health improved. The patient was in the hospital five weeks, when he went home by his own desire, and he returned to work at the expiration of three months. The pulse, which it will be remembered was 54 at the first examination, went down sometimes to 32; the average for the five weeks the patient was in the hospital was a fraction over 42 per minute.

The patient has been seen at intervals since he left the hospital. He had persevered with the iodide until five weeks ago, when, as the appetite failed, it was considered advisable to stop the drug for a time. Latterly the patient has not been so well; the old treatment has therefore been renewed. The present condition is in most respects similar to that already described, but the man is thinner and his liver is more enlarged. The pulse rate is 44, and the urine is devoid of albumen.

As regards the diagnosis in this case, I am aware that that of syphilis is open to question, though the history is fairly conclusive of that disease. The points in favour of a gumma in the neighbourhood of the vena cava causing pressure on that vessel as against a like effect by a malignant growth were—(1) Length of history, five and a half years. (2) The absence of any palpable tumour. (3) The history of the patient having had an itching sore on his penis twenty years previously, and that the treatment for it had lasted but two months. (4) If the obstruction was thrombotic, syphilis

might still be the cause. The improvement of the general health and the removal of the more urgent symptoms by anti-syphilitic treatment tends to support the syphilitic theory.

On the other hand, the possibility that a thrombus formed in the vena cava in consequence of the tardy circulation, due to the bradycardia which followed the influenza, had to be considered.

On searching the literature of the subject, as far as time would allow, I found that pressure by some growth or tumour was the cause of the obstruction in most cases. Pressure by a malignant growth in proximity to the vena cava is so obvious a cause of obstruction in that vessel that it need not detain us. Pressure by a large hydatid cyst—I have found two instances of this recorded, one in the second volume of Sir Thomas Watson's 'Lectures,' and one by Elias. Dr. D. J. Macintosh* records a case of thrombosis of the inferior vena cava following enteric fever, and Dr. J. Wilson one of thrombosis obliterating the vessel as a result of inflammation. As regards the syphilitic factor in the causation of obstruction of the inferior vena cava, I must confess there is but meagre evidence to offer. Dr. Bristowe† in his papers on 'Visceral Syphilis' reports five cases, in three of which there was obstruction of the superior vena cava. In Case 1 there was no definite history of syphilis or of any cutaneous eruption, but she had had an ulcerated sore throat of long duration shortly after her marriage twenty years previously, and her first four children were either stillborn or died at birth. Here there was a tumour in the epigastrium, and the case was at first put down as malignant disease. Later on a diagnosis of gummatous disease was made, and a satisfactory result followed specific treatment. Case 2, a man æt. 43, who had syphilis thirteen years previously, for which he had not been treated. He had exfoliation of some of the hard palate twelve years after infection. Among other signs of tertiary disease he had obstruction of the superior vena cava, with dyspnœa and dysphagia. Antisyphilitic treatment restored the patient to health, so that some dilatation of the veins was the only remaining sign of his malady. The other three cases illustrate tertiary lesions in different parts of the body, a point of interest being that in two out of them there was no history of syphilis. Mr. Hutchinson, in the twenty-eighth volume of the *Transactions* of the Pathological Society, re-

* *Trans. Glasgow Path. and Clin. Soc.*, 1891–3.
† *Lancet*, 1885, vol. i, p. 331.

marks: "I should like to ask the attention of surgeons and pathologists to the question of affections of the veins and lymphatic trunks in late constitutional syphilis. But few cases of syphilitic phlebitis have as yet been recorded; yet probably most surgeons are familiar with the fact that inflammations around varices (and even about healthy veins) are not unfrequent in the subjects of syphilis. I think also that I have seen several cases in which the thrombosis and phlebitis were attended by other conditions sufficiently peculiar to justify a belief that they were of specific origin. In some there has been great excess of inflammation, a hard mass forming in the cellular tissue and threatening to slough, much as subcutaneous gummata do. These cases are much benefited by iodide of potassium, as far as the prevention of sloughing is concerned, but the thrombotic plugging remains."

Dr. H. Mendel, in an article on "Syphilitic Phlebitis" in the *Archives générales de Médecine* for 1894, remarks that this disease had till then been very little studied, nevertheless several published observations show that the venous tissue is no more safe than any other from syphilitic infection. All the veins may be attacked, but he had only seen one case where the peripheral veins were affected, and he has only collected twelve cases owing, as he believes, to failure of recognition.

The author goes on to say that in the primary stage of syphilis several veins are affected, either at once or successively, and the condition is amenable to treatment. In the tertiary forms it confines itself to a single vein, where it produces much more serious lesions. These two varieties conform to the usual course of the disease; the early lesions are slight and more diffuse, while the later lesions affect deeper structures.

The writer refers to two cases mentioned by Langenbeck. In the first one a woman, aged fifty years, had on the right side of her neck, at the level of the cricoid cartilage, a firm and painful tumour adhering to the skin and the sterno-cleido-mastoid muscle. Cancer was diagnosed, and an operation decided upon. At the operation it was seen that the tumour had no connection with the carotid, but it adhered tightly to the external jugular vein. A portion of the vessel had to be resected in order to remove the neoplasm. The vein was invaded by an adherent thrombus. Under the microscope the tumour was found to consist of embryonic

tissue. The patient, who had denied having had syphilis, manifested tertiary lesions in the following year.

In the second case the syphilitic history was clear from the first. The patient, a woman æt. 42, had a tumour as large as a fist above Poupart's ligament on the left side. The diagnosis lay between sarcoma and cancer. At the operation the tumour was closely adherent to the vein and femoral artery. The vein was degenerated and invaded by an adherent thrombus. This patient died of pyæmia, and the author remarks that, had a correct diagnosis been made, iodide of potassium might have saved her life.

The post-mortem revealed infarcts in the lungs and amyloid degeneration of the kidneys. The femoral vein contained an adherent and decolourised thrombus, and the internal coats of the vein were friable. This change in the venous tunic extended to the external iliac vein.

In the seventeenth volume of the *St. Thomas's Hospital Reports,* Mr. Battle has contributed a valuable and exhaustive paper on the subject before us, and I desire to express my indebtedness to him. In discussing the question of prognosis, the author concludes it is "serious in all cases *except where the block is due to a gumma.*"

X.—*Remarks on Twelve Consecutive Cases treated by Operation for Perforating Gastric Ulcer.* By ARTHUR E. BARKER. *Read December* 8, 1899.

THERE is probably no *accident* to the alimentary canal which has more interest at the present moment for physicians and surgeons than that of perforation of the stomach by gastric ulcer. It is of interest for many reasons, physiological, pathological, and surgical, quite apart from the almost certainly fatal result which follows if left without operation. Of course everyone must admit that it is not a very common accident on the whole, and ought to be less so if the rules laid down by physicians for the guidance of dyspeptics were more carefully followed. But the fact remains that a considerable number of patients in the prime of life are every year lost from the rupture of gastric ulcers. I am reminded of this by a glance at my own list of cases, which shows that within the last four years I have been called upon to treat fourteen such cases of perforation, and have operated for the closure of the lesion in twelve. And it appears questionable whether the frequency of this accident will really diminish, in spite of improved methods of medical treatment. In the first place, in the great centres of population the conditions of life reacting upon the digestive system unfavourably are becoming more and more artificial, and in the second a considerable proportion of cases of perforation have not had the advantage of previous medical treatment, owing to the partial or complete *latency* of their symptoms before the actual rupture. I have been struck with this latter fact in reading over the notes of many cases. In one patient in the subjoined series under my own immediate observation for months before the accident, the condition of health appeared to be perfect up to the moment of the seizure. In others some slight indigestion was the only trouble complained of by otherwise healthy individuals. In only three was there a history of previous vomiting. In eight of the twelve cases there was certainly no previous hæmatemesis, and in only two was this symptom obtainable. Indeed, most of the cases appear to have dispensed with serious medical treatment before the final rupture, owing to the comparative latency of their symptoms.

It appears probable, then, that for a long time to come surgery will be frequently invoked for the saving of life in these terrible accidents, and it appears therefore desirable that every fragment of information in regard to them should be gathered up, and made familiar to all practitioners as far as possible.

The first need for a successful treatment of these cases is of course the *early recognition* of the lesion. If this is true for the medical management of gastric ulcer, it is doubly so for the surgical treatment when the latter has perforated. It is with this accident, of course, that I am here especially concerned. And with a view to obtaining all the information possible in regard to the symptoms and treatment of such cases, I have carefully studied all my own cases taken together, while at the same time giving, I hope, due weight to the experiences of others. Groups of consecutive cases occurring in the practice of one man, are admittedly of more value than those collected far and wide, treated by different methods, and under varying conditions.

In the subjoined table I have tabulated all the leading facts in regard to the twelve cases operated on by myself for the closure of a perforation due to gastric ulcer.

A glance at this table brings out some interesting and useful facts. Ten out of twelve cases were young females in the prime of early womanhood, and all unmarried. They all belonged to the domestic servant class. This would appear to show that they were well housed and clothed, and had at least the opportunities of healthy dieting. But one cannot help speculating as to whether their change from poorer houses and diet to surroundings in which they could indulge in varieties of unaccustomed food has not been injurious to their digestion, especially as many of them came from homes in the country to the crowded metropolis, where their opportunities for exercise in fresh air were smaller. Only two of the cases were men, one æt. 46, an artist of some repute, and the other æt. 24, a pattern-maker.

The symptoms following the actual perforation were not always the same. But the first, *i.e.* epigastric pain, was common to all. This was mostly sudden and very severe, and usually followed some distinct muscular exertion, but not invariably. In Case 5 the patient, a young and most robust-looking woman, went to bed quite well to all appearances at about 10.30 P.M., and on turning over to go to sleep felt violent pain in the abdomen at about 10.45.

Collapse as a sequel to the perforation is noted in only six of the twelve cases. It was certainly absent in four other cases, slight in one, and the remaining case was not marked enough to be specially noticed. This absence of shock with perforation in some cases is a remarkable fact, and of the utmost importance from the point of view of diagnosis. It tallies with what we all know of gunshot and stab wounds of the stomach. It is quite evident that in estimating the probability of perforation it cannot be implicitly relied upon. The shock in these cases appears to depend rather upon the amount of fluid extravasated into the abdomen and its quality as an irritant, than upon the lesion itself. In other words where the amount of escaped fluid is small, or, if large, where it is bland and unirritating, the shock is slight or may be entirely absent. In all the five cases which recovered, the last meal was either a light breakfast, or tea in the evening. And in two other cases, Nos. 1 and 8, which may be said almost to have recovered from the accident, one living fifteen, the other fourteen days, breakfast was the last meal. The worst cases among the rest which died were those where the extravasation occurred after an abundant dinner, the food taken being extravasated in a state of partial digestion.

The next thing which most materially influenced recovery was the time which had elapsed between the moment of perforation and the attempted relief by operation. In the five which recovered this time was respectively 8½, 18, 7½, 18, 6½ hours, or an average of 11⅜ hours; in the seven who died, 32, 28, 17, 3½, 8, 77, 48 hours, an average of 30½ hours. Of the latter group two nearly recovered; they were operated on at 32 and 8 hours respectively, the first having been in bed from the onset. The necessity for early diagnosis and the most prompt surgical interference possible is clearly emphasised by these figures; and those who see these cases at the outset have a very serious responsibility in rapidly formulating a diagnosis, and then in not losing a single moment until they are placed under surgical care. Some time must necessarily be lost in most cases, but this should be reduced by every means to a minimum.

In estimating the value of absence of liver dulness as a symptom of perforation, I venture to think that a good deal of caution is necessary. Many clinical observers have come to the same conclusion, but there is still perhaps too much inclination here and there to regard the sign as pathognomonic of perforation. That it is not so many abdominal cases

prove. Even in this short list there is evidence of this point. In six instances liver dulness is noted as quite gone, and in all of these much gas escaped on opening the abdomen. Besides these six cases it is noted as absent as far as the axillary line in two cases. In one of these there was no free gas in the abdomen, and in the other "very little." In another case the liver dulness is recorded as "normal," and yet on opening the abdomen there was abundance of gas. In all these cases the physical signs were worked out, and noted by several competent physicians, including usually the visiting physicians of the hospital and strangers. In three cases the notes are deficient on this point. It is plain, therefore, that though this sign, if clearly present, is valuable in most cases, it may mislead in a few if taken alone. It is also clear that liver dulness may be present in cases in which there is abundant free gas in the peritoneum from perforating gastric ulcer. In one of these cases (No. 9) there was a very large amount of gas free in the abdomen, but here there was no actual perforation. That liver dulness may also be absent in cases where there is no perforation of any viscus is also well known to all clinical observers of large experience.

The explanation of the disappearance of the liver dulness after perforation which has always suggested itself to me is this. In the first place we must suppose that the liver, a heavy organ, is retained in contact with the moveable diaphragm partly by atmospheric pressure and partly by the pressure of the abdominal muscles acting on the lower abdominal contents. It is quite clear that the ligaments of the organ could not keep it constantly applied to the diaphragm.

Now when gas escapes into the peritoneum from one of the viscera the atmospheric pressure is more or less equalised, and the suction action between liver and diaphragm is abolished, the liver sinking down by its weight as the gas finds its way above it. If the patient is vertical when the perforation takes place, as is usual, this is more likely to occur, any extravasated fluid gravitating rapidly towards the pelvis, while the gas would rise above it. If, however, as in Case 8, the patient at once be placed recumbent, and the amount of gas be small, and if at the same time the action of the abdominal muscles be very strong, on account of irritation and pain, the liver would be retained in its place, and the air would not have the same chance of getting between it and the diaphragm. Moreover the steady pressure on the stomach would tend to prevent the escape of gas from it as long as

the patient did not vomit, as was here the case. In none of the cases I have operated on for perforation was there anything like the amount of abdominal rigidity present in Case 8, to which may perhaps be attributed in part, at all events, the absence of gas in the abdomen.

The conditions found in Case 9 appear to me to be difficult to explain. On the one hand there was an enormous quantity of gas in the peritoneum, which obliterated the hepatic dulness, and on the other no perforation could be found, in spite of very free handling and pressure upon the distended stomach, and wiping away of lymph from its surface. And indeed, *after death* the distended stomach had to be completely removed from the body before the fact of an ulcer being present could be ascertained. That the ulcer present was the cause of the state of things found, I have no doubt. It was large, sharp cut at one end, and had a small fresh blood-clot at its thinnest spot. But, though tested with water and air, it did not leak, nor was there any perforation elsewhere in the intestine. My own explanation of the course of events in this case is, that the ulcerative process gradually approached the serous coat until nothing retained the contents of the stomach but the latter. At the moment at which the severe pain began, three days previous to admission, I imagine the peritoneal covering over the ulcer to have become inflamed without rupturing at all. It is well known now that while healthy bowel allows none of the organisms of fermentation to pass through its coats, diseased bowel will do so. I imagine, therefore, that over the ulcer there was an infection of the peritoneum rapidly running on to general peritonitis. The gas I suppose to have been generated within the peritoneal cavity by the saprophytic organisms which had passed through the damaged spot, probably the *B. coli*, which we know generates gas. Of course we must admit that there may have been a small pin-hole opening at first, which subsequently became closed by plastic matter. But this is hardly likely to have been the case, for at the operation the stomach was found to be distended with gas, and although it was freely handled no leakage could be produced. Besides this, after death no trace of opening could be discovered. And not only this, but the amount of gas free in the peritoneum was very great, and was *increasing rapidly;* and if there was no perforation, or if the perforation had been soldered up, whence came the rapid distension? When the patient was examined during the afternoon by Dr. Bradford,

the liver dulness was not quite gone. When he examined the abdomen on the operating table it was obviously so.

I cannot help, therefore, regarding this case as one of infection of the abdomen from an inflamed spot over a deep ulcer, with generation of gas within the latter, as one of the products of the bacterial infection, but without strictly speaking any perforation.

Again, it is well known to all surgeons that under pressure gas may pass through damaged bowel in large quantities, even where there is no perforation. This is often seen in cases of chronic obstruction for which we are doing colotomy. And, of course, the same might take place through the diseased but not perforated coats of the stomach. But in this case there was at the time of operation general septic peritonitis, and it seems more natural to suppose that with the infective organisms of the latter were the gas-producing bacteria besides.

In regard to the operative measures undertaken in these cases, they were almost identical in all. And herein lies the special point of interest of the series. They can now, as a whole, be contrasted in regard to results with any equal group operated on by other procedures. The leading points in the surgical treatment were these. The patients were in all cases wrapped up in heated wool and surrounded with hot bottles to prevent chill. The operation was carried out as rapidly as possible to diminish shock, and, for the same reason, with as much gentleness in handling the viscera as might be. In all I relied rather on sponging or mopping out the abdomen than on flushing out the peritoneum, which was not carried out in any of these cases. A median incision above the umbilicus was found adequate in eight cases. In four this was supplemented by a transverse division of the left rectus muscle in order to secure working space. No ill effect has appeared to follow the latter incision. Drainage in the flank was only provided for in one case, and did not appear, as the event showed, to have been necessary. In none of these cases did it appear at the time desirable to excise the ulcer. But looking back now at Case 8, I think it is probable that life might have been saved by this measure. It will be noticed that here there was much induration round the perforation, and consequently much difficulty in closing the latter. Nevertheless the patient did exceedingly well for eight days. He then began to bleed from the ulcer into the stomach, and died on the fourteenth day of this continuous hæmorrhage. Had the ulcer been excised, this might, perhaps, have been pre-

vented.. But it is questionable whether he would have survived the extra shock due to this addition to the operation. No autopsy was permitted.

The sutures used to close the wound were in all cases of fine, hard-twist silk, inserted in two rows, one to close the opening, and the other to support the first. Some were interrupted and latterly continuous.

The drainage through the anterior wound was in all cases provided for by wisps of iodoform gauze. Two of these went between the liver and diaphragm on either side of the falx, one was laid behind the gall-bladder, one over the line of suture, and one straight downwards behind the umbilicus. This drainage appears to have been adequate. The gauze was removed, one piece at a time, before the end of the first week, and the wound was allowed to granulate up, assisted by strapping with American plaster.

The condition found on opening the abdomen was in all cases very startling. In nearly all much free gas was present, and a large amount of fluid. This latter was almost universally distributed in all, being most abundant in the flanks and pelvis, but also in the subphrenic spaces. These latter required special care in the process of wiping. This was usually carried out by passing the hand and mop or sponge on either side of the falx, and sweeping it over the whole surface of the liver downwards and backwards. In this way much lymph and even particles of food were cleared out of this dangerous space. And I cannot help thinking that this is one of the most important details of the whole procedure, remembering the frequency of subphrenic abscess where perforation of the stomach has taken place. It is quite clear from these and allied cases that the general cavity of the peritoneum can dispose of a considerable amount of toxic substances; *for no one ever has left, or probably ever will leave it quite free from irritating matter* at the end of such an operation. And yet many patients have had uninterrupted recoveries where the cleansing of the peritoneal cavity has only been, so to speak, relatively good. But when pockets of undiluted stomach contents are left undisturbed, even in small amount, the danger is great. And in the case of the subphrenic space such fluid is very likely to be left undisturbed, unless we make it a fundamental rule in all these cases to explore it with hand and mop in every case, and assure ourselves in this way that we have left it at all events relatively, if not ideally, clean. The risks of neglect of this precaution were well illus-

trated by my first case, where, not being so impressed with its importance as I am now, I did not so carefully mop out the subphrenic spaces as I have since done. Here, although in other respects the patient was doing more than usually well for ten days, a subphrenic abscess of large size then developed, and was soon followed by pleuro-pneumonia by direct extension through the diaphragm, causing death on the fifteenth day. And Cases 7 and 12 also illustrate the same risk, though the subphrenic spaces had been specially cleaned. In Case 7 a collection of thin turbid odourless fluid was found by aspiration in the right subphrenic space on the eighteenth day, and freely opened, the patient rapidly convalescing after this; and in Case 12 there were symptoms which led several who saw the case to diagnose subphrenic abscess on the right. Exploration negatived this, and the patient recovered, these special symptoms slowly disappearing. My belief is that in this last case there was only a small dose of the irritant left behind after the mopping, which was very carefully done, and that this was not enough to cause an actual abscess, but was enough to produce basal dry pleurisy, with a little pneumonia. In Case 7 there was a little more of the irritant probably left in the space, enough to cause a moderate and limited effusion, which was opened through one of the intercostal spaces and drained successfully. It was evidently only mildly septic, and had not irritated the pleura or lung very much, as was seen in operating through the lower part of the pleura for its evacuation. All this cleaning of the subphrenic spaces required a considerable expenditure of time, but this was justified, notwithstanding the grave condition of the patient at the time.

Not infrequently the first incision and the escape of gas was followed by considerable improvement in the pulse and respiration. A knowledge of this fact ought, I venture to think, to influence us in the moment chosen for operation. It is sometimes a question whether the patient's strength is sufficient to justify any interference at all, and the surgeon is tempted to postpone the operation. But if there is much distension of the abdomen present, I think we are certainly justified at all events in making a small incision as far as the peritoneum, so as to evacuate the gas and some of the fluid. The diaphragm will now have more room to act, and both the heart and lungs being relieved of pressure may respond in the most marked manner. If they do not, the case need not be carried further; it will be probably hopeless.

In all the cases of this series the ulcer was found on the anterior aspect of the stomach, and, though in some cases very high up and towards the cardiac end, none were inaccessible to suture. In one case there was a well-marked "hour-glass" stomach and old adhesions, both adding to the difficulty of closure. No perforations or ulcers were found on the posterior aspect of the organ.

As a rule the convalescence of the five successful cases was steady if slow, after the first effects of the long operation coming upon the depressing accident of perforation had passed off.

The outline of the after-treatment may be briefly described as consisting in cautious early feeding by the mouth with easily digestible fluid food, and frequent feeding by the rectum. In the last case the patient was greatly benefited both during and after operation by the hypodermic injection of normal saline solution in quantities of from half to a whole litre at a time.

The prognosis in cases which have been operated on for perforated gastric ulcer is another point of great interest. Such patients must be carefully followed up for long periods before we can speak positively in regard to their expectancy of life. But as far as my own cases are concerned, four out of five have done well up to date over periods amounting to years. Case 2 (1895) was lost sight of about a year after the operation, but was then well and in service. Case 5 (1896) was heard of recently, well and in service. She had had threatening of gastric trouble some months after the operation; but a few days in bed, with restricted diet for a long time after, resulted in complete convalescence. Case 7 (1896) has been heard of recently. She is reported to be quite well and in service. From Case 11 (February, 1899) a letter was received a few days ago stating that she was quite well and in service, and only asking for a set of false teeth, as she could not masticate her food. Case 12 (June, 1899) has been recently examined in hospital (November, 1899). She seems quite well, but it is perhaps too soon to pronounce finally.

None of these cases appear to be the worse for the large abdominal scar, whether vertical or transverse.

The following cases are the only ones which now call for notice, my first seven having already been recorded in the *British Medical Journal*.

Case 8. *Perforated gastric ulcer; operation; death.*—On

November 24, 1896, at about 4 p.m., I was asked by Dr. Coode Adams to see a gentleman at Hampstead who had apparently ruptured a gastric ulcer at about 12.30 or 1 o'clock P.M. on the same day. The history was that the patient, an artist æt. 46, had been suffering from severe gastric disturbance for several months, for which he had been treated by Dr. Patrick Manson. He had had several attacks of vomiting, with hæmatemesis and melæna, and severe pain in the epigastrium. At the same time he was in poor health generally, and had evidence of a cavity in the apex of the right lung. At about 12.30, while exerting himself in removing some of his pictures and furniture, he was suddenly seized with violent pain in the epigastrium and sickness. At this time he was practically fasting, having had nothing but a little milk that day. Dr. Adams on being sent for found him sitting on the edge of his bed in great suffering, and very much excited. His regular attendant, Dr. Patrick Manson, had been sent for, and on his arrival the patient was thoroughly examined. There was no shock. The abdomen was found to be extremely rigid and exquisitely tender. There was nothing abnormal on percussion, but from the past history of gastric ulcer and the suddenness of the attack of pain, the diagnosis of perforation was made, and I was sent for to operate. When I arrived the patient was already under the influence of chloroform, which he had clamoured for as the only means of relief, a quarter and a sixteenth of a grain of morphia having failed to ease his pain. I found the abdomen still rigid, resonant all over, not distended. The liver dulness was *normal*. Pulse 100; temperature 100° in the rectum. Though I had some doubt as to the diagnosis, I felt with the other medical men that an exploratory operation ought to be done at once, and as the house was quite dismantled, and all the furniture was in a van at the door, I felt that the quickest means for securing the requisites for operation was to move him at once to University College Hospital. When laid on the operating table and under chloroform at 8.30, eight hours after the onset of pain, the patient was examined by Dr. Manson, Dr. Bradford, and myself, and the liver dulness was present to a normal extent, nor was there anything unusual about the abdomen except great rigidity. On reaching the peritoneum by a median incision above the umbilicus, I made a puncture in the membrane where it bulged, and there was immediately a puff of gas. When widely opened much fluid was found in the peritoneum, thin, serous, with flakes of white lymph in it.

The left rectus was now divided transversely, and the stomach was drawn down and the liver up. Much lymph lay upon the surface of the former, and a little to the right of the centre of the anterior surface a perforation about the size of a goose-quill, in the middle of a mass of induration the size of a five-shilling bit, was found. Through this milky fluid was escaping with bubbles of gas.

The stomach was now emptied by pressure, as in other cases, and was drawn down for suture of the perforation. This traction had a most unfavourable effect on the pulse, which became very weak and intermittent, and ether was injected subcutaneously. Considerable traction was necessary to bring the whole within reach, and great difficulty was experienced in turning in the thickened edges and suturing them together. Silk was used, and nine stitches were placed in the first row, several of them cutting their way through the inflamed tissues more or less. A second row of eight stitches was then inserted, which appeared to close the aperture satisfactorily. After the formal sponging of the whole abdominal cavity, as in other cases, I laid a long strip of omentum over the line of suture, and over this a strip of iodoform gauze, with another strip on either side reaching to each lobe of the liver. The wound was then closed by stout silk sutures, except at its upper angle, where the gauze drain lay.

The pulse was fairly good at the end of the operation, which had lasted one hour and twenty-three minutes, and next morning the patient was much better. Pulse 128, temp. 98°.

The further history of the case was one of steady progress, though sickness lasted for the first two days. The bowels acted freely on the sixth day after operation, when the abdominal condition appeared quite satisfactory. Part of the gauze drains were removed on this day, and again on the eighth day, when there was some abdominal pain.

On the thirteenth day melæna was noticed, with pain in abdomen. Bright red blood was vomited frequently, and patient sank on fourteenth day from hæmorrhage from the ulcer. Otherwise the state of the abdomen was all that could be wished. No post-mortem was permitted by the friends.

Case 9.—Walter H. This was a young, fairly healthy-looking man, æt. 24, who had been treated at University College Hospital as an out-patient for some time for symptoms of gastric disturbance, *i. e.* pain in the hypochondrium, sick-

ness, vomiting, and constipation. On Saturday, November 21, at 5.30 P.M., he was seized with severe pain in the belly. He went, however, to the theatre afterwards, but was unable to remain. He was given some brandy to relieve the pain. On Sunday he was no better, and passed no water; and on Monday he was in the same state, and was sick. He was admitted on Tuesday. The abdomen, when I examined him at 6 P.M., was much distended; the percussion note was clear all over, except in the right groin, where there was deficient resonance. The liver dulness was almost quite abolished, if not completely. The heart was pushed up by the distension, its strongest impulse being felt in the third interspace. His pulse was 100, temp. 101° in the rectum. On the operating table the liver dulness was totally obliterated.

At 10 P.M., seventy-six and a half hours after perforation, and immediately after the last case, I operated exactly as I had just done. On exposing the peritoneum by a median incision I punctured the latter, and immediately there was a strong rush of gas, which could be felt by the hand six inches off for about half a minute. When the cavity was freely opened a very large amount of inflammatory fluid was found among the intestines, which were greatly distended and with difficulty retained in the abdomen. This fluid was most abundant between the stomach and liver on the left side and in the pelvis. When it had been removed by sponging, a large amount of white lymph was seen lying on the stomach, but when this was wiped away *no perforation could be found.* The stomach was full of gas, and no amount of squeezing could produce any evidence of bubbling through a perforation. After a prolonged search for the latter, it was suggested that it might be found in the abdomen; but here, too, it could not be demonstrated. I then tore through the omentum and examined the posterior surface of the stomach, but this was quite healthy; nor was there any fluid in the sac of the omentum. I therefore sponged out of the peritoneal cavity large quantities of white and gelatinous lymph and serum, and partially closed the opening in the abdominal wall, leaving gauze drains as in other cases. The operation lasted one hour fifteen minutes.

The patient bore the operation extremely well, but towards morning he began to flag, and died at about 2 P.M. next day.

At the autopsy general peritonitis was found, with much gelatinous and dirty-coloured lymph. The duodenum was healthy; and it was not until the whole stomach had been

removed and slit open that the cause of the condition could be found. It was then seen that there was a large ulcer about 1½ inches from the cardiac orifice on the lesser curvature. This was about an inch long, and ⅓ inch broad. But though deeply excavated at its lower end it had *not* completely perforated, but was still closed by the peritoneal coat, which was quite smooth externally. It neither allowed the passage of water or air out of the stomach, and it was strong enough to withstand all the handling of the organ, both during the operation and at the post-mortem examination, without the least leakage.

Both these cases were alike in the fact that they were males, and had suffered for some months from severe gastric disturbance.

The first striking point of difference was that, though gas was present in the general abdominal cavity, the liver dulness was in no way lost in the first, but was totally gone in the second. Further, in the first of these two cases there was a distinct perforation, as large as in several others on which I have operated where the liver dulness was obliterated, and yet the latter was unaffected in this case. On the other hand, in the second of the two cases there was no actual perforation, and yet the amount of gas in the peritoneum was the largest have yet known in such accidents.

Case 10. *Perforated gastric ulcer; operation; death.*—Martha C., æt. 26, shop assistant, suffered from gastric pain and vomiting for a long time, and was an out-patient at St. Bartholomew's Hospital for this condition. On Saturday, July 23, 1898, was well relatively, but was seized with sudden severe pain in the abdomen. On Monday, 25th, was seen by doctor, who found temp. 100·2°; abdomen distended; bowels confined. Patient walked from cab into hospital. Pulse 144, full, strong; resp. 34; temp. 100·6°. Admitted 11.30 A.M. No collapse; movement causes pain. Abdomen slightly distended, and moves but slightly on respiration. Abdominal wall firm and resistent, and tender in epigastrium. Liver dulness present, except between parasternal line and right nipple; slight dulness in flanks, elsewhere resonant.

Operation 2 P.M., July 25, at least forty-eight hours after perforation. Median incision, and later transverse in addition; a large quantity of offensive fluid escaped. An hour-glass constriction was found two to three inches from the pylorus, and on the anterior surface of the narrowed portion was a

small perforation through which much fluid could be pressed. Round this hole there was wide-spread induration. Besides this there was a large firm cord of fibrous adhesion, as thick as a goose-quill, between the liver and the stomach, which had to be divided before the latter could be fully exposed. Between the left lobe of the liver and the diaphragm was a large collection of matter, bounded below by spleen and stomach,—in fact, a large subphrenic abscess. This was cleared as well as possible, as were also the flanks and pelvis, which likewise contained inflammatory fluid. I then with much difficulty closed the hole in the stomach with seven deep and four superficial Lembert sutures. Finally, after another formal mopping out of the abdomen, five iodoform gauze drains were left in the upper part of the abdomen as usual, and I closed the lower part of the incision. The pulse, which before the operation was 132, fell to 120 when the abdomen had been opened, but by the end of the operation had risen to 142. At 9 P.M. it was 142, and of good quality. Next day, in spite of all effort, the powers failed, and patient died about thirty-two hours after the operation.

The cause of death was purulent peritonitis. The hour-glass constriction was verified post mortem, and a drawing was made. It was further from the pylorus than was supposed during the operation.

CASE 11. *Perforated gastric ulcer; operation; recovery.*—Elizabeth B., æt. 21, domestic servant. Indigestion for twelve months; much more for last week. No hæmatemesis, but has vomited after food occasionally for last twelve months.

February 26, 1898, at 8 P.M., sudden sharp pain in epigastrium. Vomited soon after, and all night up to 12.30 P.M. on 27th; pain intense all the time. The last solid meal was at 1 P.M. on 26th, and cup of tea at 5 P.M. The illness began, therefore, seven hours after the last solid meal, which consisted of mutton, potatoes, and parsnips.

On admission at 12.30, February 27, great pain complained of. Breathing shallow. Pulse 120, temp. 102·2°. Little distension, but great rigidity and tenderness. Liver dulness gone in front, but normal in nipple line.

Operation at 2 P.M., eighteen hours after perforation. Median incision, and division transversely of left rectus. Local anæsthesia failed in deeper structures. Chloroform. No gas escaped on opening peritoneum. Liver glued by lymph to stomach and colon. On releasing it the stomach was drawn

forward, and a hole size of a goose-quill found in middle of anterior surface. After cleansing the whole abdomen with sponges and sterile gauze, the hole in stomach was closed with two rows of Lembert stitches. After a final sponging the omentum was thrown up over the stomach and the abdominal wound was nearly closed, leaving strips of iodoform gauze in various directions for drainage.

Bore the operation fairly well. Pulse 140 on removal to bed.

From this time an improvement was steady. The upper part of the abdomen was soon quiet, and except for several stitch abscesses in transverse wound there was little or no suppuration. But in the lower part of the abdomen there was distension; and on the tenth day the temperature rose to 102·8°, and the pulse to 158. The next day pulse 132, temp. 102·4°; general condition better. I now felt sure that there was some localised collection of pus deep in the pelvis, and hoped it would burst into the vagina or rectum. On March 24 there was a blood-stained discharge from the vagina with fall of temperature, which went on for some days, when mucus and pus began to come away from the rectum. With this the temperature and general condition soon improved greatly, and convalescence followed.

Case 12.—Sarah H., æt. 32, servant, lived in London for last sixteen years, and has had a very easy life. Appetite poor, and bowels constipated for last nine years, with indigestion on and off. Otherwise very healthy. Brought up ℥ij of blood eighteen months ago.

Seized with sudden pain in upper part of abdomen at 6.20 p.m. on June 30, 1899, while going upstairs, and half an hour after cup of tea; pain also felt between shoulders and across back. No vomiting then. Retching came on in the evening, the last full meal of mutton and vegetables having been taken at 2 p.m.

When admitted was in a state of profound collapse, lying on her back and complaining of great pain in the upper part of the abdomen. Temperature 100·4°; pulse 132, small and fluttering. The abdomen did not appear to be distended, but the respiration was thoracic and shallow—40 to the minute. The abdomen was hard and resistent all over, and exquisitely tender. The liver dulness was obliterated, except over a small area in the axillary line. There was also a small area of dulness in the left flank. All over the rest of the abdomen the percussion note was tympanitic.

Operation at 1 A.M., June 30, six and a half hours after the perforation. Chloroform. A median incision above the umbilicus was made, and no other. A quantity of turbid fluid and very little gas escaped. The wound was then enlarged to two inches below the navel, which was excised. The stomach was adherent to the left lobe of the liver, and when separated from the latter the perforation was found 1½ to 2 inches from the pylorus, and near the lesser curvature. The hole was ⅜ of an inch across, and was surrounded by much induration for 1 inch in every direction. When the stomach had been squeezed dry and the whole abdomen mopped out, including the pelvis and round the spleen specially, the hole was closed by a row of interrupted sutures with great difficulty, owing to the induration around it. A final mopping and partial closure of the external wound completed the operation, which had lasted one hour and ten minutes. No flushing was done, and the wound was drained with four strips of iodoform gauze. During the operation one litre of sterile normal saline solution was injected under the skin and ♏iij of Liquor Strychniæ; brandy was also given by the rectum, but none hypodermically. The saline injection was repeated under the skin at 5 A.M. to the extent of 600 c.c. The following afternoon there was great dyspnœa, and the pulse became almost imperceptible. This was met by ♏x of ether under the skin and ℥j brandy by rectum, as well as 500 c.c. normal saline under the skin with Liquor Strychniæ ♏iij every three hours. Patient vomited at 7 P.M., but her general condition was better. From this time on she improved steadily until July 2, when the stomach was washed out with brandy and hot water with relief. On July 4 a normal motion was passed. On July 6 there was a rise of fever and irritable cough. The right lower base was dull and vocal, fremitus abolished, with loss of breath-sounds. This was supposed by some who examined the patient to be due to subphrenic abscess. I was not myself satisfied on this point, as the liver was not depressed, and I had seen but little trace of lymph over the right side of the liver during the operation. In the afternoon there was much dyspnœa. The next day I explored through the seventh intercostal space with an aspirator as far as the substance of the liver, but got nothing, though I repeated the puncture twice.

The next day under chloroform I again punctured with negative result through the sixth space in the anterior axillary line and in the third space in mid-axillary line; and again on

July 8 with the same negative result. The stitches were removed this day, the wound being quite healthy. The chest symptoms remained as before, but other points remained favourable, the bowels acting naturally.

On July 10 the area of dulness was noted as less, and the breath-sounds were returning; the vocal fremitus was felt lower in the right axilla, and the cough had disappeared.

From this on there was general improvement; the chest symptoms cleared up. The patient was out of bed on July 27, and left the hospital at the end of the month practically well. She has been seen this month (November) at hospital looking very well. There is still slight deficiency of resonance over the right base, but she does not complain.

Case 11.—Elizabeth B. has also been seen lately quite well. She has been given a set of false teeth as part of the treatment of her general tendency to gastric disturbance.

Table of

No. Sex. Age. Date.	Previous and present symptoms.	Duration of perforation.	Liver dulness.	Time and quality of last meal.	Operation.
1. F. æt. 23 Dec. 1, 1895	Sudden epigastric pain, but not too severe for work, for 4 hours. First seen 28 hours after. Then collapsed. P. 110. T. 102°. R. shallow. Distension and tenderness.	32 hours	?	Breakfast just before perforation	Median incision above navel. Abdomen mopped dry. Double row of silk sutures. Wound partially closed over drainage gauze. No flushing out of cavity
2. F. æt. 20 Dec. 19, 1895	Anæmia for last year, menstruation irregular. Constipation; pain in epigastrium and dorsal region; retching, anorexia, cough. No hæmatemesis or headache. On admission not much shock. P. 120. T. in rectum 103·2°. R. shallow, 30. No distension, but wall rigid and tympanitic	8½ hours	Gone	Breakfast at 8 A.M. of bread and butter, tea, and egg. Some pain followed, but went to work. At 11.40 A.M. coughed; severe epigastric pain, with fainting and semicomatose condition. At 12.30 P.M. vomited, and again 2 P.M.	Median incision and transverse through left rectus. Fluid mopped out, especially subphrenic spaces. 7 silk sutures taking up half an inch, placed vertically. Omentum laid over them. Drain in left flank, and gauze drains in median wound. Operation 1 hour 20 minutes
3. F. æt. 23 Jan. 10, 1896	Epigastric pain for some years. No vomiting, and no hæmorrhage. 2 P.M. on 9th severe attack of epigastric pain spreading over abdomen. Repeated vomiting, but no hæmatemesis. Pale and collapsed. P. 160. R. 60. Great distension	28 hours	Gone	Dinner just before onset was abundant	Median incision. Escape of much gas, with improvement of P. and R., and return of liver dulness. Abdomen and stomach emptied. When clean, double row of silk stitches after division of fibrous band. Mopping and drainage with gauze as usual. Operation 65 minutes
4. F. æt. 17 Feb. 29, 1896	Indigestion for 6 months, marked by pain after meals. Constipation, but no vomiting or bleeding. Last motion 25th. On admission, well nourished, but collapsed. P. 90. T. 99°. R. quick and shallow, thoracic. Distension general	12 or 17 hours	Gone	Breakfast just before severe pain, induced by perforation. (?) Ate dinner at 1 P.M. At 1.30 very severe pain, and had to give up work and go to bed	Median incision, with transverse section of left rectus above navel. Abdomen mopped out *sec. artem;* stomach emptied through perforation. Silk sutures (six). Operation lasted 75 minutes

Operations.

Condition found. Size of hole. Edges of perforation.	Clinical subsequent history.	Result.	Remarks.
Much gas and fluid, with remnants of food. Perforation size of goose quill to left of middle of anterior wall	Bore operation well for 3 days. Then T. rose, but otherwise well. Bowels acted well on 10th day. Subphrenic abscess diagnosed this day. Lung symptoms set in at the last	D. 15th day	Had I had the courage to operate for the subphrenic abscess, recovery would probably have followed, as a localised abscess was found above the spleen with pleuro-pneumonia. P.M. showed subphrenic abscess, otherwise a satisfactory state of things.
Much gas and turbid fluid escaped, with return of liver dulness. Vomiting on handling the stomach Perforation ½ inch high up to left of middle. Much induration, but no adhesions. Whole abdominal cavity full of fluid, most in left flank and pelvis	Moderate shock, but not for long. Convalescence steady	R.	Writes Nov., 1899, that she is quite well, and again in service. Only complains that her teeth are bad, and asks for artificial ones.
Stomach strongly adherent to parts around, and especially by old fibrous cord, size of goose quill, to abdominal wall. Perforation about middle of anterior wall, surrounded by gristly hardness	Patient only lived 4 hours, having been in a critical state all through the operation	D. in 4 hours	There was practically no hope for this patient from the moment the condition of the interior of abdomen was seen. It was foul, with decomposed food everywhere. Death was due to collapse.
Two pints of fluid and much gas escaped, with return of liver dulness. Potatoes and greens in abdominal cavity. Perforation ½ inch diameter in anterior wall near lesser curvature	Much shock, and death in 12 hours from collapse	D.	This was a very hopeless case from the outset. P.M. showed general septic peritonitis, with collections of semi-purulent fluid. The line of suture was watertight.

Table of

No. Sex. Age. Date.	Previous and present symptoms.	Duration of perforation.	Liver dulness.	Time and quality of last meal.	Operation.
5. F. æt. 24 June 12, 1896	Robust and always healthy except for very trifling dyspepsia. Never had any signs of serious stomach disease. No bleeding. At time of perforation looked perfectly well. R. inguinal hernia. When first seen just after onset at about 11.30 p.m. only severe pain was complained of in R. inguinal region. No collapse. P. 80. T. 99·8°. No sickness; good colour. Inguinal region free.	18 hours	?	Tea and bread and butter at 8 P.M. Perforation at 10.30 P.M. after getting into bed quite well and on turning over	Regarding this as possibly a case of *reduction en masse* of R. inguinal hernia, I opened the L. inguinal region. No hernia, but much fluid and gas escaped. Then median incision above umbilicus. After careful mopping *sec. artem* 2 rows of 6 silk sutures closed the perforation, which was further covered by fold of omentum. Operation lasted just 2 hours
6. F. æt. 20 Oct. 25, 1896	Dyspepsia for a year past, otherwise healthy. At 5.30 P.M. violent pain in abdomen and collapse at once, with tenderness in epigastrium. On admission there was still collapse, hurried breathing, surface cold, pallor. P. 108. T. 100·6°	3½ hours	Gone	Dinner of beef and cabbage at 1.30 P.M. The perforation pain set in at 5.30 P.M.	Median incision above navel. Abdomen mopped dry after emptying stomach. 7 silk sutures closed the perforation, reinforced by 10 external to them. Drainage with iodoform gauze strips as usual. Operation lasted 80 minutes
7. F. æt. 27 Nov. 2, 1896	Strong healthy woman, but anæmic, with very bad teeth. Dyspepsia for years, but no hæmatemesis. Constipated usually. Was found in her room at 11 A.M. motionless, and apparently dead. Recovered by stimulant. On admission, severe pain in epigastrium. Respiration shallow. P. 80. T. 102·2°. No distension	7½ hours	Gone	Soon after breakfast	Median incision above navel. Whole cavity mopped out. Hole closed by silk sutures, 9 in first row, 8 in second. Iodoform gauze drains as usual. Operation lasted 83 minutes

Operations—continued.

Condition found. Size of hole. Edges of perforation.	Clinical subsequent history.	Result.	Remarks.
Much turbid and glairy fluid filled the whole abdomen, and gas was abundant. No food detected	Shock very severe. The patient left the table quite pulseless, and remained so for a long time. Then gradual return of pulse and warmth. Convalescence was uninterrupted. Patient has been heard of recently in good health, and in service in Norwich	R.	The difficulty of diagnosis was very great, especially as the patient was one of my own servants and particularly healthy to all appearance. She had only reduced her hernia just before going to bed and removing truss. Except pain, which was localised in the R. iliac region, there was no symptom until next morning, and then only slight fever added to pain. The first incision was clearly only an exploration on a provisional diagnosis.
Much gas and turbid fluid, mixed with green cabbage and other food. This fluid was faintly alkaline. Perforation ¼ inch in diameter. It lay near lesser curvature to left of middle. Edges clean cut, but surrounded by much induration	Comparatively little shock, which soon passed off. Next morning condition good. From this the T. and P. rose, and patient died 36 hours after the operation, with T. 106·2°	D.	General septic peritonitis was found P.M.; the only part of the abdomen found free from it being the area of operation.
Abundant effusion and gas all over abdomen, but most to left side. Perforation near lesser curvature to left of mid line, ¼ × ¼ inch in size, with very little induration around it	Bore operation well, and got on for 7 days satisfactorily. Then symptoms of R. subphrenic abscess began. On 18th day I put in an aspirator needle in 8th interspace in mid-axillary line, and at a depth of 2 inches drew off 13 oz. of thin turbid fluid. Next day I removed portion of rib at same point, and after suture of two leaves of pleura evacuated the subphrenic abscess and drained. Convalescence steady after	R.	In this case the only complication was the subphrenic abscess. After its evacuation all went well. The patient was heard of quite well 18 months later.

Table of

No. Sex. Age. Date.	Previous and present symptoms.	Duration of perforation.	Liver dulness.	Time and quality of last meal.	Operation.
8. M. æt. 46 Nov. 24, 1896	Pain in stomach on and off 9 years. Never sickness or bleeding until 10 months ago, then severe pains with hæmatemesis and melæna. At 12.30 severe pain and loss of breath. No shock. On admission, great pain: vomiting occasionally, P. 100. T. 100°. R. quick	8 hours	Normal	?	Median incision. Abdomen mopped dry. Edges of hole turned in with great difficulty. Gauze drains as usual. Operation lasted 83 minutes
9. M. æt. 24 Nov. 24, 1896	Dyspepsia 6 months, with pain after food. No vomiting; no bleeding. On Nov. 21st had dyspepsia with pain; became worse. No vomiting; no bleeding. On admission, severe pain; great distension and tenderness; fluid in abdomen	77 hours	Gone	Tea just before pain set in	Median incision. Abdomen mopped clean, and drainage as usual. Operation lasted 75 minutes
10. F. æt. 26 July 25, 1898	Chronic dyspepsia, with pain and vomiting. On 23rd was seized with sudden severe pain in abdomen. On Monday, 25th, doctor found distension and T. 100·2°, with constipation. Patient was able to walk from cab into hospital at 11 a.m. Then P. 144 full and strong. R. 34. T. 100·6°. No collapse; movement causes pain; flanks dull	48 hours at least	Present, except from parasternal line to nipple	?	Median incision, with transverse division of L. rectus above umbilicus. Thick fibrous cord divided before stomach could be drawn forwards. Cleansing of abdomen, especially of subphrenic abscess, also of pelvis and flanks. First row of 7 silk stitches, second of 4 stitches. Gauze drainage.
11. F. æt. 21 Feb. 27, 1899	Indigestion 12 months; worse last week; vomiting, but no bleeding. At 8 P.M., Feb. 26, sudden pain in epigastrium. Vomited soon after. P. 120. T. 102·2°. Only slight distension, but much rigidity and tenderness	18 hours	Gone in front but present behind nipple-line	Last solid meal at 1 P.M. on 26th, and cup of tea at 5 P.M. Dinner consisted of mutton, potatoes, and parsnips	Median incision and transverse division of rectus above umbilicus. Abdomen mopped out dry. Hole closed with 2 rows of silk sutures. Gauze drainage.

Operations—continued.

Condition found. Size of hole. Edges of perforation.	Clinical subsequent history.	Result.	Remarks.
Gas first, then fluid, mostly in loins. Perforation with wide-spread induration at small curve. A good deal of lymph	Steady improvement for 8 days. Then some abdominal pain. On 13th day there was blood in motions, and bright red blood was vomited until patient died of hæmorrhage from gastric ulcer	D. 13th day hæmorrhage	No P.M. examination permitted. There was every prospect of recovery until the hæmorrhage set in
Free escape of gas and fluid with flakes of lymph. Hollow viscera much distended. Extensive universal adhesions. No perforation could be found	Great shock, and gradual failure until death	D. 26 hours	P. M., a large ulcer found near œsophagus on anterior surface, which destroyed all coats except serous. General peritonitis was the cause of death.
Large quantity of offensive fluid escaped. Tight constriction of stomach found 3 inches from pylorus, on anterior aspect of which was a small perforation discharging stomach contents. Round this hole was wide-spread induration. Besides there was a firm fibrous cord, as thick as a goose quill, between the liver and the stomach. A large subphrenic abscess on L.	Condition was improved by operation at first, but next day powers began to flag, and death took place 36 hours after the operation	D. 36 hours after operation	The cause of death was purulent peritonitis. The constriction of the stomach was further from the pylorus than was supposed during the operation.
No gas, but much lymph glued liver, colon, and stomach together. Perforation size of goose quill in middle of anterior surface.	Improvement steady, but on 10th day there was some distension in lower part of abdomen, and T. rose to 102·8°. I felt sure there was inflammatory matter in pelvis. On March 24th this burst into vagina, and henceforth convalescence was good	R.	Except for the abscess in pelvis there was no disturbance of the convalescence. Last week I had a letter from patient saying she was quite well, and asking for a set of artificial teeth (Nov., 1898).

Table of

No. Sex. Age. Date.	Previous and present symptoms.	Duration of perforation	Liver dulness.	Time and quality of last meal.	Operation.
12. F. æt. 32 June 30, 1889	Indigestion and anorexia on and off last 9 years, otherwise healthy. Brought up 3ij of blood 18 months ago. Bad pain in stomach 6.30 P.M., also between shoulders. No vomit then, but came on later. On admission there was deep collapse. T. 100·4°; P. 132, small; R. 40. No distension, but great tenderness.	6½ hours	Gone, except in axillary line	Cup of tea half-hour before onset of pain. Dinner of mutton and vegetables 4½ hours before onset	Median incision. Stomach squeezed dry. Abdomen mopped well out. 1 row of silk sutures was introduced with much trouble, owing to induration. Gauze drainage. Operation lasted 70 minutes. Saline hypodermic injection during operation.

Operations—continued.

Condition found. Size of hole. Edges of perforation.	Clinical subsequent history.	Result.	Remarks.
Very little gas, but much lymph gluing stomach to L. lobe of liver. Perforation 1½ to two inches from pylorus near lesser curve. Hole size ¾ inch across, with much induration all round	Improvement until 7th day, when cough was noticed. Some signs of mischief at R. base were found, suggesting subphrenic abscess with dyspnœa. On 8th day I passed an aspirator through the lower interspaces towards the liver, but found nothing. Next day the same thing was done without result. Then gradual improvement and convalescence	R.	Probably the signs in R. base were due to dry pleurisy, with some consolidation. Patient seen lately quite well.

XI.—*A case of Arrested Development of the Speech Centre.* By Arthur Voelcker, M.D. *Read December* 8, 1899.

SUSAN W., æt. 7, was admitted under my care into the Hospital for Sick Children, Great Ormond Street, on November 10, 1899, suffering from inability to speak.

Family history.—The patient is the elder of two children, the other being a boy æt. 6, who is said to be quite healthy. The father's sister has a child æt. 11, who is said to be affected in a similar manner to the patient, and who had fits as a baby. There is no family history of insanity, epilepsy, alcoholism, syphilis, or rheumatism.

Personal history.—A full-term child; natural labour. Quite well till six months of age, when the child had a series of general convulsions, ascribed to dentition. The first attack lasted fifteen minutes, and the child is described as going stiff in it. On the evening of the same day the child had thirteen more fits, but the mother did not notice any unilateral distribution of the convulsions. Between this and three years of age the child seems to have had occasional fits, but since then has had none. The child walked at twelve months, but has never spoken. When one year of age the child fell on its forehead, and had an abscess at the seat of injury which lasted for six weeks, but from which no bone was discharged. At four years of age she had measles. The child has never been violent, passionate, or destructive. She has been clean in her habits, though she occasionally wets and soils her clothes. She has never been able to speak, but appears quite bright, and seems to hear perfectly.

State on admission.—Rather small for her age. Face well proportioned. Maximum circumference of the head 19·75 inches. There is a deep scar in the middle of the forehead, but no depressed bone can be felt. The roof of the palate is distinctly high and rather narrow. Movement and sensation are natural. No wasting of muscles. Gait is fair, though the child rather tends to throw her feet about in walking. Grip fair on both sides. Co-ordination good. The child is rather left-handed, preferring to pick up objects with the left hand, though she also uses the right hand at times.

No tremor of limbs. Reflexes natural. Knee-jerks equal on both sides. The child is intelligent and understands what is said to her, and obeys instructions readily. Her hearing is normal. Spontaneous speech is limited to a few sounds, the most frequently employed being "eh." Imitative speech is limited to a few sounds, such as "eh," "bee," "bye," "baa," "gee," "tick." She repeats sounds with a numerical, though not with a phonetic accuracy, thus a word repeated to her three times will be reproduced as a thrice-repeated sound, and words of more than one syllable are represented by a number of sounds corresponding to the number of syllables. When, however, she listens to a watch or clock, and is asked what it says, she does not make any attempts to say tick, tick, tick, or to make any rhythmically recurring sound, and although she can say tick she never repeats the word spontaneously or in association with the ticking of a clock, which, however, she hears perfectly.

The child can appreciate musical sounds, and when told to sing at once begins a crooning melody, which does not correspond to any recognisable tune. In chapel when music was played she kept up a humming tune all the time. It was not demonstrable that the child could recognise any tune, nor did she make any attempt to sing in harmony with a tune, or to sing any scales when these were played on the piano. When started to say the alphabet, or a series of numerals, or days of the week, or the Lord's Prayer, there is no attempt at any serial production of sounds. "Yes" and "No" are often accompanied by their usual affirmative and negative head movements. Sight is natural. She cannot recognise printed or written words, numerals or letters, nor group similar ones together. She recognises objects and pictures of objects readily and accurately, and groups similar ones together. She cannot indicate any given colour correctly, nor group similar colours together. She correctly picks out a small from a large object.

Writing.—When given a slate and pencil, she takes the pencil, usually in the left hand, and holding it with all four fingers begins to make a series of undulatory marks on the slate, passing away from the middle line of her body, *i.e.* from her right to her left hand, but this is not always the case. When she writes with the right hand the marks are made from the left to the right, *i.e.* again from the middle line outwards. The writing does not seem to be much under the influence of visual control, the child frequently looking

away from the slate. She cannot write any letter or numeral, but can sometimes make an imperfect circle or oval when told to write O. She can copy a series of oblique lines, or of circles, or of horizontal strokes, but very imperfectly, and she cannot copy alternating circles and lines. She cannot make a X, nor put a cross into a square, nor can she place dots in a circle in imitation of a face even when a similar circle has been thus treated. It is to be noted that when she makes a series of oblique strokes with the left hand towards the middle line, *i. e.* in the usual left to right sense, she not infrequently makes a stroke in the other sense, *i. e.* to the left of the previously formed stroke.

When shown a picture of a sheep she never says "ba," though when asked who says "ba," sometimes points to the picture of the sheep. When shown a picture of a horse and asked what is that, she sometimes says "gee" or "ee," and makes use of similar sounds when shown a toy horse. A toy pig does not elicit from her any sound, though she repeated when told the words "piggy wiggy," and always recognises a picture or an imitation of a pig.

There were no evidences of malformation or disease in the body, and though a soft systolic murmur was heard over the cardiac region and in the neck on admission, this has now disappeared.

Remarks.—Speech defects in children have a peculiarity from the point of view that so many of them occur before the education of the faculty of speech.

Whether we regard speech as being an hereditary property of certain portions of the brain, or merely as the acquired function of these parts, we must recognise that its development is markedly influenced by the stimuli which are received from without, by means of auditory and visual impressions; when such stimuli have never reached the speech centre on account of defects in the auditory perceptive centre or its in-going fibres, we have a condition of deaf-mutism.

Bastian points out that in the development of the power of speech the first stage consists in learning to associate particular sounds with particular mental impressions, which association at last becomes so strong as to be almost inseparable. The thing recalls the sound—the sound the "idea" of the thing.

The second stage consists in the acquisition of the power of articulating for himself the sounds which have been used as mental symbols.

In this case the first stage seems to be reached, for the particular sound we call a name is promptly associated with an idea, which in turn is associated with an external object.

Accepting the existence of an auditory and of a visual perceptive centre, and of a glosso-kinæsthetic and of a cheiro-kinæsthetic centre, as maintained by Bastian, it would appear that the in-going fibres to, and the auditory perceptive centre itself were intact, for the child can hear everything and can identify objects and pictures of objects when named.

The in-going fibres to, and the visual perceptive centre itself, are similarly intact, the child recognising objects and pictures of objects, although unable to identify letters or numerals, or to comprehend written or printed words.

The cheiro-kinæsthetic and glosso-kinæsthetic centres are, however, involved, but the degree to which they are involved is difficult to ascertain, because the child has never been educated in the matter of writing or reading.

When given a slate and pencil, the latter is grasped clumsily with either hand, though a distinct preference is shown for the *left* hand, and the child then makes a series of undulatory lines moving from her right to left, *i. e.* away from the median line of the body. With the right hand the movements are in a similar sense, *i. e.* outwards, but are then from left to right.

The fact that we write from left to right is probably partly due to inheritance, and partly to the fact that movements with the hands are more free away from, than towards the trunk. Thus, as we educate the right hand unduly, our method of writing is from left to right; but when we consider how the movements we make with the right are represented in the left hand, we see that abduction of the right limb resulting in a left to right movement is represented by abduction of the *left, i. e.* right to left movement, and again it is easy to demonstrate the greater ease with which movements towards and away from the trunk can be made simultaneously, than movements resulting in a maintenance of parallelism of the limbs. It is much easier to describe a circle clock-wise with the right, and counter clock-wise with the left at the same time, than clock-wise with both hands. This is well seen if we attempt to do this rapidly.

Now this child has evidently had no training in writing, for she does not form a single letter spontaneously, yet her right hand movements are outwards, and her left hand movements are also outwards, and would, if she had learned to write,

constitute mirror-writing. This I think tends to show that the movements in mirror-writing are the representation in the left hand of the movements in the right, and that the impressions which pass to the fingers are the result of kinæsthetic, and not visual impressions. This would perhaps lend support to Elder's view, that there is only one special centre for writing movements, and that in the *left* hemisphere and in connection with this I can call to mind a case of embolic right hemiplegia, in which the earliest attempts at writing with the left hand were made in "mirror-writing," though afterwards the patient soon acquired the power of performing ordinary writing with his left hand.

The limited ability to copy in this case is interesting, only the simplest forms—straight lines, circles and undulations,—being correctly reproduced. This indicates a defect in the visual perceptive centre, or its outgoing fibres to the cheiro-kinæsthetic centre. The recognition of objects and pictures of objects shows that the ingoing fibres to, and the visual perceptive centre are healthy, so that the defect must be situated in the cheiro-kinæsthetic centre, or in the fibres passing to this from the visual perceptive centre.

The want of any previous education in writing or drawing makes it impossible to say how much is the want of education, and how much is due to the absence or disease of these centres; we know the readiness with which these are educated varies much in different individuals, and in this case I should expect to find that the child could be taught to copy without much difficulty, because she is capable already of copying a few elementary forms.

It would be interesting to know how those races whose writing differs from ours in the direction in which they write behave in the process of learning to write, or when the subjects of a right-sided hemiplegia. We ought to expect a mirror-writing in these cases if Elder's view is correct.

With regard to the function of speech, the condition of the glosso-kinæsthetic centre seems to be much the same as the cheiro-kinæsthetic centre, for while spontaneous speech is limited to a few sounds, which we might compare to the undulatory movements initiated by the cheiro-kinæsthetic centre, associational speech is almost absent, and imitative speech is limited to simple words like "ba," "gee," "by," comparable to the straight lines and circles of the cheiro-kinæsthetic centre.

Dr. Bastian has pointed out that the fact that speech and

writing are so frequently involved together in typical cases of aphasia is due partly to the proximity of the glosso—and the cheiro-kinæsthetic centres, and perhaps not less to the proximity of the two sets of commissural fibres connecting these centres to the auditory and visual word centres respectively, and although he does not accept as proved Exner's localisation, he shows very good reasons for believing in the existence of a cheiro-kinæsthetic centre. This case can, however, not throw much light on this point, for the parellelism of affection of the two centres might either be interpreted as an evidence of their identity, or as an evidence of a similar process occurring in neighbouring centres.

The site of disease would thus appear to be in the second and third left frontal convolutions at their posterior part.

With regard to the nature of the lesion, the question arises whether we have a destruction or merely a retardation of development of these centres and their commissures.

Dr. Bastian has recorded the case of a boy who never spoke till he was six years of age, and who had had fits in the first two years of his life. When six years of age, under the influence of emotion, he suddenly exclaimed "What a pity," and then relapsed into his condition of inability to speak till fourteen days later, when he started speaking, and "speedily became most loquacious." Dr. Bastian also refers to similar cases in his work on aphasia, and argues that speech has now become a truly automatic act for human beings; such untaught speech, as shown in the boy he refers to, is dependent on the normal development of the auditory sense and intelligence, but inhibited by some condition of the connections between the auditory perceptive centre, and the glosso-kinæsthetic centre, and it is possible that some such condition may be present in this case.

Against the view that there is any gross lesion of the central area is the absence of any evidence of hemiplegia or diplegia, and the fact that children who become the subjects of loss of speech from disease of the left hemisphere as a rule quickly recover the power of speech; so that unless we accept a symmetrical lesion as an explanation, and are prepared to account for the absence of any paralysis of the face or tongue or arm, we must seek the explanation in some arrested development of the posterior part of the third and second frontal convolutions on one or both sides.

Heredity would point to the probability of the left side being affected, and would explain why the right centres were

unprepared to take on the duties of the left, the right and left centres being in imperfect commissural connection.

With regard to prognosis this seems favourable, for since the child has been under observation very distinct improvement has taken place, and the case is not one of idiocy.

Treatment.—The late Dr. Hadden has recorded some cases of speech defect similar to those described by Drs. Hale White and Golding-Bird, and by them named idioglossia, in which the children used a language peculiar to themselves, and in whom he obtained excellent results by isolation and instruction in lip language.

The case now under consideration, however, is not one of idioglossia, and yet I think that instruction in lip language offers the best chance of opening up communications between the visual perceptive and the glosso-kinæsthetic centres, and thus of awakening the latter centre into activity.

In conclusion I should like to express my thanks to my friend Dr. King, of Stratton, who kindly sent the case to me, and to my colleague, Dr. Penrose, who not only lent me a bed for the case, but has also assisted me with his observations and suggestions.

XII.—*Two cases of Successful Primary Resection of Gangrenous Small Intestine during Herniotomy.*

By J. HUTCHINSON, JUN. *Read January* 26, 1900.

CASE 1.—A single woman æt. 40, was admitted into Mary Ward of the London Hospital on March 2, 1893, on account of a strangulated femoral hernia on the left side. She had known of the hernia for two years previously. It was reducible until February 27, 1893, when it became larger, harder, and quite irreducible. At the same time she experienced much pain in the groin and the abdomen; she had vomited for three days before admission, but apparently the vomit was not "fæcal" at any time.

On admission patient looked ill and anxious, her pulse was rapid (120 per minute) and wiry, her tongue covered with white fur, the abdomen not distended. There was a swelling about the size of a hen's egg in the left groin, just below Poupart's ligament and external to the spine of pubis; this tumour was irreducible and presented no impulse on coughing, the percussion note was dull, the skin over it was hot and a little reddened. An incision about three inches long was made over the neck of the sac, the superficial tissues being divided until the sac was reached. This was opened up in the usual manner, and was found to contain six or eight drachms of reddish serum and a knuckle of small intestine, which was of a dull purplish red colour with streaks of yellow lymph on its surface; the constriction was divided upwards and inwards, and the gut drawn out a little way, when it was found to be intensely congested for some little distance beyond the constricted part. It was then decided to open the abdomen in the middle line and resect the gut. An incision two and a half inches in length was made from a point an inch below the umbilicus. The congested portion of the gut was carefully drawn out, and it was seen that about four inches of gut were acutely inflamed, sodden, and flaccid. Five inches of gut were excised and the ends united by a double row of silk sutures. The gut above and below this portion was carefully emptied by pressure of its contents, which were prevented from returning by digital compression during the progress of the operation.

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... ...was a man, æt. 29, admitted int... ... ...December, 1896, with a strang... ... ...It was impossible to ascertain ... ...existed, as the man was a ... ...knowledge of a word of English. At the operat... ... ...quite flaccid, and comme... ... ...eight inches. After division ... ...was drawn upwards ... ...median abdominal incision ... ...the operation. The peritoneal c... ... ...to the ... ...ten inches we... ... ...sected by Ma... ... ...ular enterorrhaphy by

Now the sac of the hernia was ligatured at the femoral opening and cut off, and the external wound closed. Both the abdominal wound and that in the groin were dressed with iodoform and Tillman's dressing.

During the whole operation the A.C.E. mixture and ether were used. The pulse never failed, but maintained its strength, nor did the patient's appearance alter.

Progress of case.—Patient had some pain during that night, when the only hypodermic of morphia (gr. $\frac{1}{6}$) was administered. Nausea was experienced, but no vomiting. For the first thirty-six hours only a little ice and some hot water in small quantities were administered by the mouth. Then milk was taken, commencing with 4 drachms per hour, and increasing gradually. No solid food of any description was given for a fortnight, when patient began with soft bread and milk. The temperature on the second and third days rose to 100° at 8 A.M. and 4 P.M. respectively, but remained at normal after the fourth day. Bowels did not move till the twelfth day, when an ordinary enema produced a copious result. The patient was kept in bed for three and a half weeks. At no time was there any distension of the abdomen or any sign of peritonitis, nor indeed did the patient complain of pain at any time except the night after the operation. She left the hospital about six weeks after admission to go to the country.

October, 1899.—During the six and a half years which have elapsed since the operation the patient has been repeatedly under observation; there has been slight bulging of the median scar and return of the femoral hernia, easily controlled by a combination of belt and truss. Otherwise she has continued in excellent health, and has not had the slightest indication of narrowing of the intestines.

CASE 2.—The patient was a man æt. 29, admitted into the London Hospital in November, 1896, with a strangulated right inguinal hernia. It was impossible to ascertain how long the strangulation had existed, as the man was a Pole and understood not a word of English. At the operation I found the small intestine black, quite flaccid, and commencing to be gangrenous for some eight inches. After division of the constriction, the gangrenous loop was drawn upwards from the hernial sac through a median abdominal incision, care being taken to prevent infection of the peritoneal cavity. The gut being evidently quite hopeless, ten inches were resected by Maunsell's method (circular enterorrhaphy by inter-

DESCRIPTION OF PLATE I.

To illustrate Mr. Hutchinson's Case of Resection of the Small Intestine.

a. Scar of longitudinal incision through which the intussuscepted part was drawn for suturing.

b. Transverse scar at the site of the resection.

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rupted silk stitches traversing all the coats, the suturing being done after producing an intussusception through a longitudinal incision in the convexity of the bowel). After all the sutures had been tied, the knots facing the lumen of the gut, the artificial intussusception was withdrawn, and the longitudinal incision sewn up in the usual manner.

The patient was nourished *per rectum* for five days. The two abdominal wounds healed by first intention, and there were no complications of any sort.

In September, 1899, three years after the resection, he suffered from intestinal trouble again, this time from inflammation of the colon, and died in the Metropolitan Free Hospital. My friend Mr. Harold Barnard, under whose care the patient came, kindly procured the specimen for me. It shows not the slightest trace of narrowing of the gut; the fine silk sutures have all disappeared, though their former situation can be partly recognised by minute scars in the peritoneum. There were no adhesions to the scar of the abdominal wound (such adhesions are, I believe, quite exceptional if the peritoneum be sutured separately). It need hardly be said that the colitis from which the man died had nothing whatever to do with the previous operation.

When gangrenous gut is found in a hernia the mortality following conservative treatment is admittedly so high as to justify almost any attempt to improve it. This is true, whether the intestine be simply left to slough *in situ*, or whether an artificial anus (which will practically always be in the small intestine, and sometimes near the upper end) be made. The death-rate in either case is at least 80 per cent. It is not easy to collect now many cases of this kind, but of twelve instances which I found in recent hospital records (*i.e.* where an artificial anus was made, the damaged loop being fixed to the skin) eleven died. In the only one of the twelve that survived (a patient under the care of my colleague, Mr. F. S. Eve) a successful resection was carried out some few weeks after the formation of the artificial anus.

Acute peritonitis spreading upwards generally carries off the patient, and it is well known that at the time of operation in these grave cases of herniotomy the wall of the gut is usually inflamed or ulcerated beyond the actual margin of the gangrenous area. When resection is done it is essential to divide the intestine where its wall is healthy, or at least not obviously inflamed. Thus, for example, in one of the cases of resection with sutures, "although six inches had been re-

moved, it was found that not enough had been taken, the adjacent intestine being on the verge of gangrene, and having caused peritonitis." Probably excision of ten or even twenty inches of intestine is attended with no greater risk than that of two, and certainly with less if it alone ensures healthy edges to bring together. Starting, then, with the admitted fact that to leave gangrenous gut in a hernia is almost invariably fatal, the question resolves itself into what method of primary resection affords the patient the best chance of recovery. I believe that there is a general impression that the introduction of Murphy's button has been followed by a great improvement, and that it is the best method to adopt. The only statistics which to my mind can be relied on are those obtained from hospital registers.

Mr. Kendal Franks in 1893 published a valuable series of cases of primary resection performed by immediate suture, in which the mortality worked out at a trifle under 50 per cent. It must be remembered, however, that this list was made up of cases published by a large number of surgeons, who would have been unusually candid if they had included all their fatal cases.

The following record includes thirty-one cases, a much smaller number than that given by Mr. Franks; nearly all of them were operated on since the date of his paper, and none of them were included in it. They comprise fifteen cases of resection with the use of Murphy's button, and sixteen by simple enterorrhaphy.

Beyond the fact that they were all operated on by hospital surgeons there has been no sort of selection. The post-mortem record, whenever available, has been procured, so as to test the cause of failure. I think these thirty-one cases may be accepted as affording as fair a test as can be obtained of resection with sutures and resection with the use of Murphy's button, at any rate when done for the condition of gangrenous small intestine in a hernia. The result is surprising, and it would seem decisive. Of the sixteen cases treated by one or other method of suture six, that is about 40 per cent., recovered. Of the fifteen cases in which Murphy's button was used only one (7 per cent.) recovered. Nor does the comparison cease here. Apart from one or two cases in each list which died from shock or peritonitis within a few hours of the operation, the fatal cases treated by suture survived on an average twice as long as those in which Murphy's button was used. In fact of the ten fatal cases after suture, it may

be said three would have recovered but for accidental complications; in one (operated on by me) six inches had been excised by Maunsell's method, and the patient's death six days later was due to obstruction of another loop of intestine by an omental band within the abdomen. The union of the divided ends was firm and there was no peritonitis. In the second of the three (also my case) the patient had unfortunately phthisis, with constant cough; she lived ten days after the enterorrhaphy, and at the post-mortem "the union was good, but there was a small abscess close to one stitch." In the third case, in which the patient lived three days, "the union was perfect and there was no peritonitis," but two strictures were found in the small intestine which had nothing to do with the hernia.

End to end anastomosis by means of sutures undoubtedly takes more time than by Murphy's button, and this is thought to be a very important factor. There is, however, no evidence that any one of the ten fatal cases died from shock due to a prolonged operation, and the only one in which the anæsthetic was considered to have anything to do with the death, was one in which Murphy's button was used (this patient had bronchitis, and survived the operation only twenty-four hours). It is noteworthy that nearly all the suture cases survived at least two or three days, and of the fatal ones, with the already noted exception of three, in which death was due to accidental complications, giving way of one or two stitches or inflammation around them was the chief cause of failure. Comparing those cases treated by Murphy's button, the failure of the method to procure good union was much more marked; in no less than 50 per cent. of those ending fatally there was either leakage opposite or acute inflammation around the button, in fact in only two of the fourteen fatal cases was it noted at the post-mortem that the union was good ("union practically watertight," "closure by button good"). In neither of these two cases had the patients survived more than forty-eight hours. The conclusion may be drawn that careful suturing, although it takes more time, is a more efficient method than the use of Murphy's button, and therefore gives the patient a much greater chance of recovery. How can it benefit a patient to have an operation completed in half an hour instead of an hour, if his chance of recovery is thereby diminished five or six fold?

To turn to the successful cases, the only one which recovered after the use of Murphy's button was operated on by my

colleague Mr. E. Hurry Fenwick; three inches of gangrenous gut were excised, and the patient made an uninterrupted recovery, the button being passed on the twenty-fifth day. Of the six successful cases treated by suture, there was one in which Maunsell's method was used. The length of intestine excised was respectively three, five, seven, eight, and ten inches. Curiously in the case in which only three inches were removed the operation lasted the longest time, nearly two and a half hours. The only complication to note was a fæcal fistula in one case, which formed on the seventh day and healed up entirely after a week or two.

This was no doubt due to the giving way of one stitch, in fact a careful study of the fatal cases convinces one that this is the usual cause of failure, and probably Maunsell's method, in which a great number of fine silk stitches are passed through all the coats, with especial care paid to the mesenteric attachment, is admitted by many to be the most secure method.

As regards mechanical aids to suturing, one can only say that rubber bags introduced into the intestine are of little or no value, besides being cumbrous, and that the forceps devised by Dr. Laplace are very ingenious.

In none of the six successful cases was any form of bone bobbin used, and in only one or two of the fatal cases; the deduction may be made that surgeons generally do not consider them of much importance.

It is to be feared that in spite of ever-improving medical education, now and then strangulated herniæ will be treated by poulticing or castor oil, and gangrenous gut will continue to be met with as a result.

Elaborate apparatus for performing resection will very likely not be at hand at the time of operation, and it is therefore satisfactory to know that careful suturing appears still to give the best prospect of success. There is little doubt that the resection should be performed through a median abdominal incision. In a recent case of femoral hernia in a woman, the knuckle of gut was quite flaccid, and its appearance was so suspicious of gangrene that I feared resection would be necessary. When, however, the loop was drawn out through a median incision, the circulation in the damaged area and the tone of the intestinal wall recovered, and I returned the gut and closed both wounds with a good result.

The chief reason why the stitches sometimes give way at the end of a few days, is the contamination by decomposing intestinal contents. Supposing the two ends have been

accurately stitched together, would not the patient's chance be increased by giving a saline aperient in order to secure a contracted and empty condition of the gut? Once this has been obtained, feeding could be carried out chiefly *per rectum* until union is firm.

Since this paper was written two valuable articles on the same subject have appeared, the first by Mr. Arthur E. Barker, in the *British Medical Journal,* December 23, 1899, the second by Mr. W. P. Montgomery, in the same journal for January 13, 1900. Both papers record two successful cases of primary enterectomy for gangrenous hernia. In all these four successful cases direct suturing without artificial aids was employed, and both surgeons agree in rejecting the use of Murphy's button for the purpose.

It may be of interest to give the results of a hundred and forty consecutive cases of operation for strangulated hernia which I have treated in the London Hospital, and from which the cases already quoted have been drawn.

It is noteworthy that amongst these one hundred and forty cases were no less than twenty-one umbilical herniæ, in which the prognosis when strangulation occurs is notoriously very grave.

Of sixty-five operations for strangulated *inguinal* hernia eleven (17 per cent.) proved fatal; of fifty-four operations for strangulated *femoral* hernia 14 (26 per cent.) proved fatal; and of twenty-one operations for strangulated *umbilical* hernia nine deaths (45 per cent.) resulted.

The total mortality in a hundred and forty cases worked out at 25 per cent. In the great majority of those thirty-four fatal cases, death was due to the acutely inflamed or gangrenous condition of the hernial contents.

TABLE I.—*Perforated or Gangrenous Gut in Hernia treated by other methods than resection. Eleven cases; eleven deaths.*

1. Woman æt. 64. Small perforation of intestine in femoral hernia, bowel opened and sewn to skin. Death next day. *Post-mortem.*—Extravasation of fæces into peritoneal cavity. (St. Bartholomew's Hospital, 1896.)

2. Woman æt. 74. Gangrene of gut in femoral hernia. Enterotomy with glass tube. Death on third day. (St. Bartholomew's Hospital.)

3. Woman æt. 59. Strangulated femoral hernia for four days. Perforation found, fluid fæces gushing out. Lembert's sutures applied; abdomen washed out through supra-pubic incision. Death eighteen hours later.

4. Woman æt. 64. Very collapsed with strangulated femoral hernia. Gut found gangrenous; opened and fixed to the skin. Died three days later. (Middlesex Hospital, 1897.)

5. Woman æt. 42. Small intestine (gangrenous opposite site of constriction) sutured to wound and opened on second day. On *twelfth day* an attempt was made to close the artificial anus, which caused the patient's death in a few hours. (Middlesex Hospital, 1897.)

6. Woman æt. 55. Femoral hernia; strangulated for four days. The omentum and intestine contained in the sac were found to be sloughing. Enterotomy. Death an hour or two later.

7. James W., æt. 21. Inguinal hernia. Gut (gangrenous) stitched to wound. Death four hours later. (Own case).

8. Mary S., æt. 58. Right femoral hernia; strangulated seven days. Artificial anus made. Death from exhaustion. (Own case).

9. Alfred T., æt. 20. Right inguinal hernia; strangulated three days. Intestine gangrenous. Artificial anus. Death the same day. (Own case).

10. Eliza S., æt. 56. Gangrenous intestine in femoral hernia. Gut stitched to wound. Death on third day. (Own case).

11. Eliza T. Umbilical hernia; strangulated three days. Gut in doubtful condition; returned and drainage-tube left in wound. Fæcal fistula formed four days later. Death on twentieth day after operation. (Own case).

TABLE II.—*Cases of Gangrenous Intestine in Hernia treated by Enterectomy with Murphy's Button.*

1. Woman æt. 40. Femoral hernia; strangulated three days. Ten inches of small intestine resected, and the ends

joined by Murphy's button. The patient died next day. *Post-mortem* "showed that the two halves of the button had not been accurately approximated, and that mucous membrane protruded between them." (St. Bartholomew's Hospital, 1896.)

2. Woman æt. 29. Right femoral hernia; strangulated seven days. The part strangulated was gangrenous and perforated four inches above the ileo-cæcal valve. Median laparotomy. Four inches of intestine removed (owing to the proximity of the ileo-cæcal valve special difficulty was experienced in using the Murphy's button). Extra stitches were applied round the bottom—continuous and interrupted. The hernial sac was excised. Temperature rose steadily to 102°, and patient died forty-eight hours after the operation. *Post-mortem.*—"General peritonitis with effusion of lymph—most marked at the ileo-cæcal region. At the seat of operation the gut had almost joined, there being two small gaps where union was not complete, but through which there had apparently been no extravasation of fæcal matter." (London Hospital. Own case).

3. Woman æt. 42. Strangulated umbilical hernia; treated for three days by fomentations under a general practitioner's directions. Eleven inches of gangrenous small intestine resected *in situ* (ten feet from the duodenum); Murphy's button used. Temperature rose to 102°, and patient died of acute peritonitis twenty-four hours later. *Post-mortem.*—Small amount of lymph around the junction; bowel much congested near the button. (Own case.)

4. Woman æt. 43. Strangulated right femoral hernia. Herniotomy; gut found gangrenous and perforated; median section; four inches resected. Death two days later. *Post-mortem.*—"Leakage around the button with acute peritonitis."

5. Man æt. 33. Right inguinal hernia; strangulation symptoms only slight, hence taxis was tried. At operation eight inches of gut were found to be gangrenous and were resected *in situ.* After fixing the button a small rent was found in the intestine close to it; this was sutured and the loop left in the sac. Death on the fourth day after operation from acute peritonitis.

6. Woman æt. 52. Irreducible omental umbilical hernia.

Not strangulated, but causing trouble. Operation. Omentum excised. Small intestine beneath found tightly constricted by inflammatory adhesions and almost ulcerated through. Resection; Murphy's button introduced, and a few extra Lembert's sutures applied. Patient lived a fortnight. *Post-mortem.*—Disease of aortic valves (extensive). The intestine had given way and leaked freely. General peritonitis.

7. Woman æt. 58. Right femoral hernia; strangulated eight days. Mr. E. Hurry Fenwick operated; finding about one and a half inches of small intestine gangrenous, he resected three inches *in situ*, using a Murphy's button. Gut returned. Drainage of abdomen. No complications. The button was passed *per anum* on the twenty-fifth day, and the sinus in the groin healed before her discharge. (The last four cases were in the London Hospital in 1896).

8. A man, æt. 52, with strangulated right inguinal hernia. He had had the hernia for fourteen years, and had been ill in bed for six weeks prior to admission. There was found to be an adherent appendix with suppuration around it in the hernial sac, and it was necessary to excise part of the cæcum. A Murphy's button was used. The patient died the day after operation from collapse and bronchitis. It was thought the anæsthetic given (ether) was partly responsible. (London Hospital, 1898).

9. Woman æt. 42. Strangulated right femoral hernia (Richter's) at the junction of the jejunum and ileum. Strangulated eight days. Peritonitis and abscess found at time of operation. Abdomen washed out and drained. Patient died fifteen hours after the operation.

10. A woman with strangulated femoral hernia of seven days' duration. The intestine was found to be gangrenous and perforated. Patient lived three days after the operation. *Post-mortem.*—"At the seat of operation the gut had almost joined, there being only two small gaps where union was not complete, but through which there had apparently been no extravasation." Nevertheless the patient died of acute peritonitis starting from the resection area.

11. Woman æt. 68. Strangulated right femoral hernia. The intestine was found to be perforated. Abdominal section, etc. Patient only lived five hours afterwards.

12. Woman æt. 47. Strangulated right femoral hernia. Herniotomy, followed by median abdominal section and resection of the gut; the intestine was tightly stretched over the button, and the loop was therefore fixed by suture close to the abdominal wound. Much collapse, relieved by brandy and strychnia injections. Death twenty hours after operation. Post-mortem.—Union "practically watertight." (The last four cases were in the London Hospital in 1895.)

13. In this case, a woman æt. 28, ten inches of small intestine were excised with the aid of Murphy's button. Death occurred two days later. At the post-mortem union was said to be good, though it was obviously too early to judge of this.

14. A woman æt. 60, with umbilical hernia. Part of the large and small intestine were excised, Murphy's button being used for both. Death occurred soon afterwards, but no notes are available.

Total: Fourteen cases with one recovery = 7 per cent.

TABLE III.—*Cases treated by Immediate Suture, without the use of Murphy's Button.*

1. A woman æt. 49. Left femoral hernia, strangulated for eight days and treated during the whole of this time by fomentations. The hernia, the size of a walnut, contained a little omentum and a knuckle of small intestine, which had perforated and from which fæcal matter was oozing. Median laparotomy. Excision of several inches of small intestine, by simple suturing—double row of silk sutures. Death six hours after operation from acute septic peritonitis, which had started evidently before the operation. (Own case.)

2. A man æt. 54. Umbilical hernia for twenty years, the size of an orange, had given him no trouble until three days before admission. Sac inflamed, small intestine gangrenous. Resection of six inches by means of Maunsell's method. Temperature continued normal or subnormal until sixth day after operation, when he died. Two days after the operation he commenced to vomit, and the sickness continued until his death. This was found to be due to persistence of intestinal obstruction due to an omental band. The relations of the

omentum to the sac were complex; one band had been divided, but another remained, and the small intestine above it was distended. There was no peritonitis nor leaking, but at the mesenteric margin of the resection edge the union was not perfect. It is probable that this patient would have recovered but for the persistent obstruction. (Own case.)

3. Woman æt. 50, phthisical. Strangulated femoral hernia (Richter's). Sac much indurated, gut black with ashy slough. Median abdominal section, loop drawn out and resected. Double row of silk sutures used, Lembert's externally. The resected part showed ulceration of mucous membrane almost through all coats. Patient lived ten days with a temperature constantly 100° to 102°, and had much dyspnœa and some cough. At the post-mortem a small abscess was found close to the site of union, which had given way opposite one stitch. General acute peritonitis. (Own case, London Hospital, 1895.)

4. Woman æt. 55. Right femoral hernia strangulated three days; admitted in collapse. Herniotomy, loop of small intestine found to be gangrenous and perforated. Median abdominal section. Four inches of small intestine resected, double row of silk sutures. Patient went on well for three days, then the temperature rose and she died on the sixth day.

At the post-mortem local peritonitis was present around one stitch, otherwise the union was good. (Own case, London Hospital, 1894.)

5. A woman æt. 50. Right femoral hernia strangulated three days. I found the intestine gangrenous and resected six inches. Circular enterorrhaphy through supra-pubic incision. Death from collapse two days later. There was no post-mortem, but it is probable that the union had given way. (Own case.)

6. Mary H., æt. 40. Femoral hernia; five inches of small intestine excised. Patient in good health six years later. (Own case, details given previously.)

7. Male æt. 29. Inguinal hernia; ten inches of small intestine excised. Recovery. Specimen obtained three years later. Union perfect. (Own case, details given previously.)

8. Woman æt. 41. Strangulated left femoral hernia; both gut and omentum were gangrenous. Supra-pubic inci-

sion; resection of three inches only by means of interrupted silk sutures. Enterotomy then done four inches above the sutured part, a drainage-tube left in. The wound broke down, and patient died on tenth day. "All the intestinal sutures had cut out, the two cut ends of the intestine opening freely into the peritoneal cavity, which contained much pus." (*St. Bartholomew's Hospital Reports.* Statistical Tables, 1897, p. 210.)

9. A woman æt. 44. Strangulated ventral hernia. She was much exhausted by the frequent vomiting. Three inches of small intestine were resected, and the cut ends united by simple silk sutures. The operation lasted two hours and twenty minutes. The patient made an excellent recovery, and left the hospital twenty-seven days later. (St. Thomas's Hospital.)

10. A woman æt. 35. Strangulated femoral hernia five days. Resection of three inches by circular double enterorrhaphy. Died on third day after rise of temperature to 104°. "Sutures were perfect; no peritonitis; resection portion lay ten inches from cæcum; three fibrous strictures of small gut were found." (St. Thomas's Hospital.)

11. Woman æt. 51. Strangulation four days. Resection of eight inches; circular enterorrhaphy. Perfect recovery after formation of fæcal abscess on seventh day, the fistula shortly closing, so that it could only have been a small leakage.

12. Man æt. 37. Femoral hernia. Eighteen centimetres of small intestine were resected by simple suture method. Recovery with stitch abscess at site of radical cure. Silk sutures used.

13. Woman æt. 47. Femoral hernia strangulated only twenty-six hours. The hernia was of the Richter form, and probably not much of the intestine was excised; but the amount is not certain. Good recovery.

14. Woman æt. 47. Femoral hernia strangulated seven days. Sixteen centimetres resected by simple suture method. Died. The post-mortem showed that whilst the union was firm (the patient died on the second day), not enough intestine had been removed, the adjoining part being on the verge of gangrene, and having caused peritonitis.

15. Woman æt. 56. Femoral hernia strangulated only twenty-six hours. The hernia was of the Richter form, and probably not much of the intestine was excised, but the amount is not certain. Good recovery.

The last five cases are from Franz. ("Herniologische Beobachtungen," *Deutsche Zeit. für Chirurgie,* March, 1899, p. 93.)

Total fifteen cases with seven recoveries = 46 per cent.

XIII.—*A case of Bullet Wound of the Abdomen, with Four Wounds of Small Intestine and Two of the Mesentery: Operation: Recovery.* By A. Pearce Gould, M.S. *Read January* 26, 1900.

THE subject of this injury was a robust young German, A. L., æt. 20, who on October 30, 1899, was playing with a revolver in a dormitory shared by him and several other waiters of a restaurant. One of the chambers of this revolver happened to be loaded, and its contents were discharged across a narrow table into the person of A. L., who was then wearing only his night-shirt. A doctor was very quickly brought, who found A. L. in great pain, and happily gave him a hypodermic injection of morphia, and then brought him to the Middlesex Hospital, where he was placed under my care.

I saw him about an hour after the accident. He was lying quietly on his back, was pale, and was still in some pain. His pulse was 96, rather weak but regular, his breathing was easy, and his abdominal walls moved with respiration. Two inches below the navel and an inch to the left of the middle line was a roundish, slightly contused wound, from which blood was slowly trickling.

There was no distension of the abdomen, nor was it anywhere unduly resonant; the liver dulness was not obliterated; I could not detect any dulness in either flank. He had not been sick. There was no paralysis. On passing a catheter I drew off 6 oz. of clear normal urine.

An anæsthetic (gas, ether, and then chloroform) was given, and after sterilising the skin of the abdomen I made an incision for an inch above and below the skin wound and found a hole through the left rectus abdominis to the outer side of the skin wound, and the finger passed through this muscular rent into the peritoneal cavity. I therefore prolonged my incision upwards and downwards to about 6 inches in all, so as to have plenty of room for the subsequent steps of the operation. On opening the abdomen blood at once escaped, but neither gas nor intestinal contents were detected.

The small intestine was then carefully examined, and very quickly two perforating wounds were found, apparently in the

jejunum—one near the free border, the other almost opposite, close to the mesentery. A third perforating wound was found some foot or so lower down, and beyond that again a fourth wound of serous and muscular coats only was found. The wounds measured about half an inch across, the mucous membrane protruded through the perforating ones, but there was no visible escape of intestinal matter from either of them. The perforating wounds were closed by a few points of suture in the mucous membrane, and a Halstead suture through all the coats except the mucous one. The non-perforating wound was closed by a Halstead suture only. While examining the intestine I found two holes in the mesentery, in one of which there was a vein bleeding freely; this was tied, and the wounds were closed with two points of suture. The rest of the intestine, the mesentery, and omentum, were examined, and neither wound nor bruise was found. The intestines as drawn out were protected by hot sponges. It was noteworthy that no peristalsis was excited by the manipulation. The bowel was neither distended nor congested. Blood-clots were wiped from the peritoneum, and when all was clean and dry the intestine was returned and the abdominal incision was closed by three rows of sutures, and a dressing of cyanide gauze was held in place by a binder tightly adjusted. Fine silk that had been sterilised by boiling was used for all the sutures, and these were passed into the bowel and mesentery by round sewing needles.

The whole operation occupied an hour and a half. At its close the lad's extremities were rather cold, but his pulse was only 108, his temp. was 99·4°. He was placed in bed with several hot bottles, and a hypodermic injection of ¼ gr. of morphia was given.

The patient slept a good deal through the night, and when I saw him at 10 o'clock in the morning he was warm and comfortable; except for thirst had no pain. The abdomen moved with respiration, was not tender, and was not distended. I ordered him to have ℥j of hot water every hour. In the afternoon a pint of warm water was injected into the rectum and retained. Urine was drawn off twice by catheter. In the evening he passed flatus freely.

On November 1 the water enema was repeated; on November 2 he took 1 oz. of milk and 1 oz. of water every hour; his pulse was 84.

It is unnecessary to give the detailed notes of the case during convalescence. It will be sufficient to say that his

temperature was normal throughout, reaching 99° on one evening (November 2). The pulse rate on and after November 3 was between 70 and 80. The urine had to be drawn off by catheter until November 5. His bowels were opened by enema on November 6, and about every other day afterwards. He was kept on purely liquid diet—water, milk, and coffee—until November 6, when he was allowed a little bread and butter; on November 15 minced chicken was given him, and a few days later meat.

The wound healed by first intention; he got up four weeks after the operation, and left the hospital apparently quite well on December 4.

Nothing was seen or felt of the bullet on the night of the operation; subsequently we felt and saw it by X rays lying in the left buttock, which it must have reached by perforating the pelvis from within. Here the bullet caused no inconvenience, and the patient refused to have it removed.

Remarks.—I have thought this case worthy of record because it seems to illustrate several points of importance in gunshot wounds of the abdomen.

1. The lesions produced by a single small bullet were multiple—four wounds of the small intestine, and two of the mesentery.

2. There were no symptoms by which those grave lesions could be recognised.

3. In the absence of all special symptoms it was necessary to make an exploration (*a*) to determine whether the bullet had perforated into the abdominal cavity, and if so (*b*) whether it had injured any viscus or vessel, in which case (*c*) steps could be taken to treat such injury or injuries.

4. The value of an immediate hypodermic injection of morphia, not only in relieving pain, promoting rest, and lessening shock, but also in arresting intestinal peristalsis and averting fouling of the peritoneum with intestinal contents. The inactive resting intestine was in the most favourable state for intestinal suture.

5. The position and the extraction of the bullet were of no importance.

The possible multiplicity of abdominal lesions is a difficulty which besets the practitioner in other cases than those of gunshot wounds. For example, I remember as a student seeing a well-known surgeon open the abdomen in a case of internal strangulation; a band tightly nipping a loop of bowel was quickly found and divided, and the wound was closed. To

the disappointment of all the man's symptoms continued, and he died. At the autopsy a second loop of the intestine, strangled by a second band, was found.

Three or four years ago I was called to see a gentleman suffering from intestinal obstruction, which had gradually become complete with great abdominal distension. A malignant stricture of the rectum was within easy reach of the finger, and it seemed to be quite clear that a left colotomy would relieve the obstruction. At the operation a distended sigmoid flexure was opened, but there was not that very free escape of liquid fæces that I had anticipated. This was explained very shortly after when a second malignant stricture of the descending colon was felt through the artificial anus. Such experiences are no doubt rare, but to have multiple lesions in stabs and bullet-wounds of the abdomen is quite common; and the operator must bear this in mind, and carefully examine every viscus within the region of the injury.

It is an embarrassing fact that serious injuries of abdominal viscera may give rise to no correspondingly marked symptoms. In this case there was nothing to indicate that the intestine had been wounded, or that a mesenteric vein was bleeding. Had the rule of waiting for symptoms been followed no doubt we should have had the signs of internal hæmorrhage and increasing dulness in the lower parts of the abdomen, soon followed by those of intense infective peritonitis. Even then an operation *might* have been successful, but the conditions would have been terribly unfavourable. It is comparatively easy to anticipate and prevent peritonitis, but it is a most difficult matter to arrest it when once the extensive and complex serous membrane has been widely infected. It would, therefore, seem to be the surgeon's duty to make a careful exploration of the peritoneal cavity and abdominal viscera in every case of penetrating wound, and in those cases of contusion of the abdomen in which the force of the blow renders visceral lesion reasonably probable.

Of recent years a good deal has been said against the exhibition of opium or morphia in abdominal diseases and injuries, and especially in cases where surgical interference may become necessary, chiefly because it "masks symptoms," to some extent also on account of the constipation it causes. While agreeing in the main with these views, yet I feel sure that opium or morphia may be used to great advantage under certain circumstances in abdominal lesions.

I would submit that the case I now record is a striking

illustration of one of the uses. The morphia that was given to this man within a very short time of his injury not only relieved pain and anxiety and lessened shock, but it arrested intestinal peristalsis, and thereby postponed fouling of the peritoneum by intestinal contents, and gave time for the patient's transfer to the hospital and for my operation to be completed before this dire result of his injury was produced. The success or failure of the operation depended, first, upon whether it was completed before peritoneal infection occurred, and, secondly, whether the closure of the wounds led to such an exact sealing of the peritoneum that no subsequent infection would occur. I believe that the initial dose of morphia did much to secure the first of these favourable conditions. It was remarkable to me how pale and how absolutely devoid of all peristalsis the intestines were, and also how absolutely unfouled the peritoneum was, even immediately around the perforations of the intestine. The dose of morphia soon after the operation was given to lessen shock and to secure restfulness and sleep. There is no drug so valuable for the treatment of shock as morphia.

No words are necessary to support my statement, that the bullet having come to rest in the patient's body had most probably become a harmless mass of metal, and therefore neither prolonged search to find it nor effort to remove it was called for.

XIV.—*A case of Fatal Hæmopneumothorax of Unexplained Origin.* By H. D. Rolleston, M.D. *Read February* 9, 1900.

A THIN boy, æt. 21, was admitted to St. George's Hospital on November 13, 1899, at 7.30 P.M.

His previous history showed that he had suffered from some symptoms of dyspepsia, but had never had any hæmoptysis, cough, or expectoration. There was no history of any traumatism.

On November 11 and 12 the patient had some diarrhœa and abdominal pain, and at 4 P.M. on November 12 he was suddenly seized with intense pain in the right hypochondrium, which radiated to the right shoulder and umbilicus. I happened to be in the hospital when he was admitted on the evening of the following day, twenty-five hours after the acute onset, and found him extremely collapsed, very anæmic, perspiring, and with a pulse of 160, running in character and irregular. The abdomen was resistent and did not move well; the liver dulness was normal.

It was thought that this condition might be due to general peritonitis after perforation of a duodenal ulcer. He was stimulated by hot enemata containing brandy, and strychnine was given hypodermically, with idea of getting him into a fit condition for laparotomy. He was sick shortly after this, bringing up some greenish fluid. An hour after admission he was kindly seen by my colleague, Mr. Turner, in consultation, but the patient's condition had in no way improved, and it was clear that no exploratory operation could be performed inasmuch as death seemed imminent. He was given morphine one third grain *sub cutem*, and ordered to be fed *per rectum*.

Next day, November 14, I was rather surprised to find that he was still alive, and was, in fact, better. Pulse 144; respirations 24.

The abdomen had altered much from the condition noted on the previous evening; the liver dulness had disappeared, and there was a resonant projection in the right hypochondrium below the ribs, over which cavernous breathing, a marked bell note, and metallic tinkling were heard; the

right side of the chest and abdomen moved little with respiration, while the left moved normally; the heart's apex was displaced outwards one inch outside the nipple line in the fifth space. The signs were those of a pneumothorax, but having regard to the history of dyspepsia and the extreme collapse I was inclined to think it might be a subphrenic collection of air and fluid, the result of a perforated duodenal ulcer. In the evening his temperature went up to 101·4°.

November 15.—The abdomen moved almost equally on both sides; there was hyper-resonance over the right side of the chest in front with bell note and metallic tinkling. The localised projection in the right hypochondrium simulating a subphrenic gaseous collection had gone. The pulse was 128, respirations 44, and temperature 100·8°.

At my request, after consultation with his surgical colleagues, Mr. Turner inserted the trocar of an aspirator into the hyper-resonant sixth interspace in the right anterior axillary line: a tube attached to the trocar was brought up under water. Although the chest was hyper-resonant, dark blood only came out. As it appeared possible that the trocar had passed into the liver and drawn off blood from a large branch of the portal vein, it was withdrawn, and again introduced in the fifth right interspace in the parasternal line. Air then came bubbling out at high pressure from the tube under water; as the air escaped the heart moved somewhat inwards: the air was followed by dark blood, which also came out at considerable pressure. The blood was like that withdrawn from the first puncture. The trocar was then taken out. In the evening the temperature went up to 101·8°.

November 16.—The patient had a rather restless night. The signs of pneumothorax remained much the same.

November 17.—Much the same.

November 18.—Developed copious bronchitic sounds over the left lung. Took food by the mouth.

November 19.—Considerable respiratory distress developed, and on the following day (November 20) he became cyanosed, and died from exhaustion and the effects of bronchitis of the left lung.

The *autopsy* was performed twenty-five hours after death by Dr. Ll. Powell. There was no sign of decomposition or of post-mortem emphysema. There was hardly any subcutaneous fat. The right side of the chest was opened under water in successive interspaces, with the result that dark fluid blood without any air came out. The abdomen was perfectly

healthy; the kidneys and liver, like the rest of the body, being pale, but free from any structural change.

The thorax.—The right pleura contained sixty ounces of dark fluid blood, with a few ounces of recent blood-clot. There were no pleural adhesions on either side. After removing the blood from the right pleura the right lung was found completely collapsed. Before removing or displacing the lungs a tube was introduced into the trachea, and air was driven into the lungs, the right pleura being filled with water. This was done in order to find out whether there was any rupture of the lung. No escape of air took place. The larynx, trachea, lungs, and heart were carefully removed *en bloc,* and the lungs were minutely examined externally; there was adherent blood-clot on the surface of the right lung. Microscopic sections made after hardening of the surface of the lung with the adherent blood-clot proved the absence of any inflammation of the pleura or of tuberculosis. The adherent fibrin, therefore, was not secondary to pleurisy. The blood-clot adherent to the lung was stripped off nearly the entire surface of the pleura, but no rupture was found, and inflation of the lungs under water was again performed, but without any leakage of air occurring. Water was injected into the right pulmonary artery, but without any leakage from the surface of the right lung revealing itself. The lungs were cut up but were found to be perfectly healthy, and no sign of tubercle could be found; there was no consolidation or pulmonary apoplexy; the bronchi were not blood-stained. The bronchial glands were normal.

The superior and inferior venæ cavæ were found to be healthy, and the right azygos vein was examined with a similar result.

The pulmonary vessels were opened and the intercostal arteries were examined, but no source for the blood or for the air removed during life could be found.

The trocar punctures had entered the pleura, and had not damaged the lung, the diaphragm, or the liver. The intercostal vessels were quite normal, and there was nothing wrong with the parietal pleura. Nothing was noticed to be amiss with the internal mammary artery on either side.

The œsophagus was carefully examined and found to be normal.

The heart was somewhat pale, but was otherwise healthy; there was no fatty degeneration of the myocardium.

The aorta was in a condition of hypoplasia; its walls were

thin and its lumen admitted the little finger. Microscopic sections of the carotid on one side, made by Dr. O. F. F. Grünbaum, showed no disease though the walls were thin.

There were no hæmorrhages elsewhere in the body.

Abdomen.—The viscera were quite healthy. The liver, 36 ounces, was pale, as were the kidneys (4 ounces each). The spleen, 4½ ounces, was somewhat wrinkled. The stomach, duodenum, and intestines were normal.

The right knee-joint was opened, but nothing abnormal was found. This was done on the possible chance that the patient might have been a "bleeder."

Remarks.—Cases of hæmothorax pure and simple are rare, and when they do occur are usually due to traumatism or rupture of an aneurysm. More commonly, pleural effusions mixed with a certain amount of blood, and depending on tubercle, traumatism, or malignant disease, are met with, but these are not strictly speaking the same as a hæmothorax. In this case no source for the blood could be found. This suggests a blood condition such as hæmophilia, but the trocar punctures did not bleed; there was no history of excessive bleeding, and the knee-joint that was examined showed none of the arthritic lesions of hæmophilia. There were no signs of scurvy. The blood in the pleural cavity was not examined bacteriologically, but the absence of acute inflammatory changes in the pleura render it most improbable that it was the result of a very acute pyococcal infection, such as the case of extensive hæmorrhage under the gluteus maximus recorded by Mr. Raymond Johnson,* where the cause of the hæmorrhage appeared to be an infective periostitis of the femur due to the *Staphylococcus pyogenes aureus.*

From time to time cases of hæmothorax not due to any manifest cause have been recorded, but recovery has often taken place, so that the opportunities of investigating the actual anatomical condition are even rarer. A very remarkable case has recently been recorded by Cheeseman and Ely,† in which the right pleura was tapped no less than twenty-two times, and blood drawn off on each occasion. This condition was cured by the injection of a watery solution of iodine, but hæmothorax on the left side almost immediately followed; this was tapped seven times and then cured by injection as before. Seven months later a hæmorrhagic peritoneal effusion occurred;

* R. Johnson, *Trans. Path. Soc.*, vol. xlvii, p. 32.

† Cheeseman and Ely, *American Journal of the Medical Sciences*, August, 1899, p. 162.

during the five following years this was tapped no less than forty-three times, and then underwent spontaneous cure. The authors suggest that the condition is pachypleuritis hæmorrhagica, on the analogy of pachymeningitis hæmorrhagica; but this explanation will not accord with the facts of my case.

In this case there were marked signs of air in the pleural cavity, and air was drawn off during life, and the condition must be described as a hæmopneumothorax—a rare condition—although no air was found in the pleura at the autopsy.

The association of air and blood of course suggests that there was a rupture of the lung which subsequently became closed; but, as I have already stated, the most minute examination of the lung brought no evidence in favour of this hypothesis.

The question arises whether the air could have been derived from the blood. It is theoretically possible that the blood having been first extravasated into the pleura, irregular respiratory movements produced some degree of negative intra-pleural pressure, and that as a result the gases of the blood came off.

This hypothesis seems to me more probable than that the gas was due to infection with the *Bacillus aërogenes capsulatus* or with *Bacillus coli*, inasmuch as no gas formations in the body or gas in the pleura were found after death.

Another curious point was the prominence of the signs of gas on the pleural cavity not only before, but also after paracentesis, and the absence at the post-mortem. Possibly it was re-absorbed by the blood after death.

XV.—*A case of a Rapidly Fatal Hæmopneumothorax, apparently due to the rupture of an Emphysematous Bulla.* By G. Newton Pitt, M.D. *Read February* 3, 1900.

WILLIAM E., æt. 18. He had always enjoyed perfect health, and had not had any illness since childhood, but was slightly anæmic. On September 15, 1897, he had a sore throat, vomited three times, and his bowels were opened twice. He stopped in bed for one day, but returned to his work on the next. He was quite well on Sunday the 19th, and went out at 4 P.M. for a walk, but soon returned on account of pain in his right shoulder and chest, which gradually increased. He took no supper, but at 9 P.M. went to bed. He scarcely slept, as the pain steadily increased and became extremely severe; about 4 A.M. he aroused his mother, but Dr. Stokes of Highbury was not called in to see him until 8 A.M., when he found him mortally ill, quite cold and pulseless. He had vomited two or three times during the night. When I saw him, in consultation with Dr. Stokes, at 3 P.M., he had rallied somewhat, but was profoundly anæmic; there was no pulse at the wrist, the skin was bedewed with a cold sweat, the extremities were cold; he was quite sensible, and was not restless. The abdomen was not tender; it was retracted, but otherwise normal. On percussion the right chest was tympanitic in front as far back as the anterior axillary line, behind which it gave a dull note, and posteriorly it was absolutely dull. In front, there was a bell note, with a *bruit d'airain,* and distant tubular breathing; the voice sounds were diminished in front, and absent behind. The heart was pushed over to the left, so that the impulse was two inches to the left of the nipple; the heart rate was 150. Obviously he had a pneumothorax, and there was probably also fluid in the pleura, causing the dulness which we noticed. We concluded that he had a pneumothorax, together with an accumulation of pus or sero-pus; and assumed that the history was not quite correct, that his illness dated back more than twenty-four hours, but that he had made light of his symptoms. His distress was so extreme, it was clear that unless relief could be afforded, he could not long survive. In

order to relieve the great distension we passed a large-sized Southey's tube into the sixth space in the right axilla, intending to fasten it in, and allow the air and fluid to gradually escape into a large antiseptic dressing applied over it. This treatment is generally most satisfactory for pyopneumothorax, as it relieves the abnormal pressure; and allows the air, if it passes into the pleura from the lung with inspiration, to be again extruded into the dressing with expiration. To our surprise some ounces of blood came away, but after a short time the tube became blocked by a clot. The tube was then re-inserted further forwards, when both air and blood bubbled out; the pain had been increased, and no relief had been afforded. We concluded that hæmorrhage was still taking place, and, as the tube again became blocked, it was removed and not re-inserted, as it appeared probable that a fall of the interpleural pressure to normal might diminish the slight chance there was that the hæmorrhage would cease.

It was therefore decided to defer any attempt to further relieve the tension until the next morning; he died, however, about 8.30 P.M. the same evening.

At the inspection about eight pints of fluid blood, and enough clot to fill the hands three times, together with a considerable amount of air under great tension, were found to occupy the right pleura. The right lung was tightly compressed, and the left base slightly so posteriorly. The lungs were absolutely healthy, with the exception of a small, projecting, emphysematous bulla, about half an inch across, near the right apex, the wall of which was imperfect; and attached to this was a fibrous band, of the thickness of a knitting-needle, which had been torn across. The blood may have come from a vessel in this adhesion, but no aneurysmal pouch or obviously patent vessel could be seen. The specimen was reserved in order that the vessel might be injected later, but as so often happens it was thrown away by mistake. Careful search was made at the inspection for injuries or lacerations, but none were found. There was no trace of any tubercle, either old or recent. The thorax, heart, aorta, and diaphragm were free from any perforations or from inflammation. Permission was only obtained to examine the thorax, but no cause beyond that mentioned could be detected.

Hæmopneumothorax is an extremely rare lesion; there is no reference to it in the Index-Catalogue of the United States Library, nor is it discussed in any of the standard treatises on medicine in either English, French or German, with the

exception of a reference to it in Fraentzel's article on Hæmatothorax in *Ziemssen's Cyclopædia*. He also refers to a case, of which no details are given, from the oft-quoted but always unseen Thesis of Saussier, in which a hæmatothorax is said to have led to a pneumothorax, and this may possibly have been a traumatic case. Undoubtedly, hæmopneumothorax may result from extensive laceration of the lung by a stab, bullet wound, or fracture of several ribs, or as the result of a crush of the thorax, even without fracture; or a lesser injury associated with a laceration of an intercostal or internal mammary artery, or of one of the veins of the thorax, may suffice. As a matter of fact, however, but few such cases have been put on record, as they have not illustrated any unexpected pathological incidents. A search through literature reveals nothing similar to the present case, and, with a view of attempting to throw some light upon the matter, I propose to summarise what is known as to the causes which have led to the presence of either blood or air in the pleura. Occasionally there is some obscurity as to the cause, when one of them has been detected in the pleuræ of an apparently healthy patient, but the discussion of even these does not suffice to clear up the present case.

Hæmothorax.

Causation.—When the literature of hæmothorax is examined it is seen that the cases may be divided into two classes,—those in which there has been a free exudation of blood into the pleura, and those in which the fluid, whether serum or pus, was merely *blood-stained*. Putting on one side the traumatic cases, in which blood has been extravasated, in the majority of the others the fluid has been merely blood-stained; and the number of the recorded cases of extravasation of any quantity of blood into the pleura, independently of traumatism and malignant disease, is small.

From this confusion some of the published lists give quite an erroneous idea of the causation. Thus Blumenthal has collected fifty-five published cases of non-traumatic hæmothorax, which he has classified as follows:

1. Tubercle	19
2. Secondary to other diseases	15
3. Primary	9
4. Malignant disease of the pleura	12

An examination of the notes shows that there was an effusion of blood in only three of the so-called primary cases, and in five of the malignant; and that in almost all the others the fluid was only stained with blood.

Of the three primary cases one was a foul empyema, a second was associated with phthisis, and was probably tuberculous. There only remains one case, which was recorded by Begin, in which there had been a post-mortem, when an effusion of blood with a vascular condition of the pleura was found. One case by Margueritte occurred in a patient with chronic hepatic disease.

The causes may be classified as follows:

1. *Traumatic.*—Trousseau and Leblanc made experimental wounds on the lungs of horses, and found that if one of the main arteries of the lungs or the vena cava were injured death rapidly ensued. When the vessel corresponded to one of the secondary bronchi, the lung was compressed by the extravasated blood, and the result of the pressure was to only gradually kill by heart failure. When the vessel damaged was smaller the laceration quickly filled up with clot, and the blood extravasating along the adjacent lung tissue formed a pulmonary apoplexy. If the animal survived forty-eight to seventy-two hours, lymph matted up the pleural surface of the wound, so that a nail-shaped mass of blood and fibrin was formed.

Panas also found that when one of the large vessels at the root of the lung is damaged the animal quickly dies with dyspnœa. When hæmothorax has been compatible with life, the vessel damaged was not more than the third order in size. When an animal dies within two to eight minutes the vena cava or one of the large vessels at the root of the lung has been damaged. A stab more than two centimetres long will readily close with clot. Polaillon has reported a case in which the hæmorrhage came from a diaphragmatic vein.

The hæmorrhage not infrequently comes from an intercostal or internal mammary artery; in a case of Hawkins's this was the source, from erosion due to a carious rib. When the effusion of blood is large, but has not been fatal within a few minutes, it has not originated from the main vessels at the root of the lung, nor does such often arise from a stab or puncture, as small openings close up with clot.

When a child's chest is crushed and the lung is lacerated, the hæmorrhage may be profuse, but even with severe laceration, hæmorrhage is not marked unless an important vessel

is damaged; when the lung is punctured by the fractured end of a rib, this will not be the case.

2. *Malignant disease of the pleura.*—A blood-stained effusion is not uncommon, but it is rare for the effusion to consist mainly of blood.

3. *Tubercle.*—Both acute and chronic effusions are sometimes blood-stained; more rarely, however, a considerable amount of blood may be effused in a tuberculous pleurisy; and still more rarely the blood from an eroded pulmonary artery in a cavity may in the final struggle burst into the pleura.

4. *Infective diseases.*—Blood-stained effusions have been recorded in connection with the following diseases:—pneumonia, variola, measles, typhoid, malaria, jaundice, and dysentery; but an effusion of any appreciable amount of blood is most exceptional. More cases have been recorded in connection with pneumonia than with other diseases.

5. *Degenerate blood or vessels.*—Cirrhosis of the liver, granular kidneys, and degenerate vessels, as in cases recorded by Sir W. Broadbent and Coupland.

There are certainly a considerable number of cases which do not belong to any of the preceding groups, and as to the pathology of which there may be differences of opinion. Boulay, Bourges, and other authors refer to cases of *hæmorrhagic pleurisy*, and compare them to the subdural hæmorrhages and to hæmorrhages into the tunica vaginalis, and suggest that in many cases chronic alcoholism is a factor, producing a chronic pleurisy, in which the adhesions develop imperfect capillaries which lacerate readily.

Occasionally bloody fluid, with or without clot, has been found in the pleura, the causation of which has remained *obscure*.

The following case was cleared up when it was shown that the source of the blood was due to the development of *leptothrix*, and probably some of the other cases may have owned a similar origin.

Dr. Freyhan records the case of a young man, æt. 22, who had previously enjoyed good health, in whom there was an effusion. with constitutional disturbance, of 500 c.c. of bloody fluid into the left pleura, which required paracentesis. The expectoration was blood-stained and abundant. It was free from tubercle bacilli and pneumococci, and was not offensive. At the end of a fortnight it was noticed that there was a strong odour of yeast in his breath, and on

examination the sputum was found to contain numbers of plugs of fungus, consisting of mycelium and conidia, which were identified as leptothrix. The right lung in turn became affected, the fungus was present in the sputum for three weeks; gradually the sputum became more purulent and the patient completely recovered.

Does the Blood which is extravasated into the Pleura remain Fluid, or does it always Clot?

Experimentally Charles Nelaton found that when blood was injected into the pleura of a dog some bloody serum was always found after some time in the opposite pleura. This is very suggestive, and would show that when there is blood breaking up in the vessels it may be expected sometimes that blood-stained serum will be found in the pleura. For example, when 600 grammes of blood were injected into the left pleura of a dog, four days later there were 875 grammes of serum together with 275 of clot on that side, and 275 grammes of serum in the right pleura. In that interval the pleural lymphatics had become clogged, and additional serum had accumulated in both the pleuræ.

Trousseau, on the other hand, found, when experimenting with much smaller quantities in proportion, in horses, that the fluid quickly absorbed, that clotting took place within five minutes after the blood had been injected into the pleura, and that it did not act as an irritant. When 200 c.c. were injected into the pleura no trace remained after forty-eight hours; and when from 500 to 1000 grammes were injected a clot amounting to about one fifth remained after three days. The induction of rapid clotting, however, may in some cases have been induced by the absence of any aseptic precautions; because Pagenstecher's experiments showed that the pleuræ, similar to the blood-vessels, are capable of keeping contained blood fluid for a long time; and that the production of clotting is, similarly to that of thrombosis, often started by a lesion of the walls. Two hours after blood was injected it was almost always fluid; after six there was a fluid resembling blood in appearance, but it would not clot. No inflammatory changes were induced. The pleuræ, therefore, can keep the blood fluid, but not unaltered. A considerable portion of the fluid is absorbed. In his experiments blood in the pleura was absorbed within a fortnight, but Wintrich concluded that a

traumatic hæmothorax required six to twenty-eight days to absorb.

Lesdos, in the *Manuel de Médecine* of Duplay and Reclus, raises the question as to whether the blood behaves differently in man from what it does in animals, and concludes that further experiments are still necessary. His conclusion, in which he is also supported by his professors, is that the blood often remains fluid in the pleura.

The amount of the effusion, the condition of the pleura, and the cause, are all factors in determining the rate of absorption of the fluid.

Clinically, also, the condition of the contents has also varied. The blood has been sometimes fluid, but in traumatic cases more often it has clotted, but almost invariably there has been some accumulation of serum as well. Sometimes the fluid has clotted after removal, as in a case recorded by Lereboullet of a man of eighteen, from whose chest 950 grammes of bloody fluid, which clotted after its removal, were withdrawn.

Pneumothorax.

The causes of pneumothorax may be classified as follows:

1. *Punctured wounds of the thorax. Laceration of the lung or bronchi by compression of the chest or by fractured ribs.* A punctured wound of the lung will not produce hæmothorax unless an important vessel is injured, but the smallest puncture may be sufficient to produce a pneumothorax.
2. *Perforation of tuberculous cavities.* This is by far the most common cause.
3. *Empyema opening into the lung.*
4. *Abscess of the chest wall opening inwards.*
5. *Perforating gastric ulcer, with a subdiaphragmatic abscess, burrowing upwards.*
6. *Gangrene of the lung.*
7. *Acute abscess of the lung.*
8. *Bronchiectasis.*
9. *Pulmonary apoplexy with gangrene.*
10. *Hydatid.*
11. *Rupture of an emphysematous bulla.* This has occurred in men apparently perfectly healthy, and has often ended in recovery. Dr. Hall and Dr. Goodhart have especially drawn attention to these cases. Fraentzel also has pointed out that the rupture may occur as the result of violent physical effort,

in one case as the result of attempting to lift a heavy cask, and in a case of Finlay's after attempting to stoop down backwards.

12. *Tracheotomy.* This occurs when the operation does not relieve the obstruction, or when the tube has been accidentally pushed down in the connective tissue to one side of the trachea, or even when the incision is a very low one. An emphysema takes place into the connective tissue of the anterior mediastinum, and then a rupture occurs, allowing the air to pass into the pleura.

13. *Perforation of the œsophagus.*

Pathology of the Present Case.

The following would therefore appear to be all the causes that can produce an extravasation of both blood and air into a pleura.

1. Traumatism. The air could come from a punctured wound of the chest wall, or from injury to the lung tissue, and the blood from one of the arteries of the chest wall or from the lung.

2. Phthisis. The blood in the pleura could arise from an eroded pulmonary artery, or from a tuberculous pleurisy.

3. Gangrene, abscess of the lung, or a pulmonary apoplexy with necrosis.

4. Sloughing malignant disease of the lung.

5. Hydatid.

6. Ruptured emphysematous bulla, with hæmorrhage from a ruptured vessel, or from some abnormal blood condition, or from traumatism.

In this case the first five conditions were definitely excluded. There is no question that the pneumothorax was due to the rupture of an emphysematous vesicle, but the source of the hæmorrhage was not so clearly proved. Nothing could be found to account for it, beyond the torn fibrous band, in which possibly there was a patent end to a vessel. It was noted at the time, however, that the amount of blood was very large to have arisen from so small a vessel.

The blood which was drawn off while the boy was alive clotted; and therefore it was clear that in this instance, clotting had not taken place at once.

Experience has shown that when the hæmorrhage in the pleura is profuse, it is generally wiser to wait and not to draw

off the blood at once. This was the treatment we adopted, as it did not appear at the time that drawing off the blood would have saved the boy.

Appendix.

After bringing this case before this Society, I examined the records of the fatal cases at Guy's Hospital of pneumothorax and of hæmothorax between the years 1885 and 1898, and am indebted to my colleagues for permission to refer to them.

During this period there have been twenty-eight inspections in which a pneumothorax was found, the causation of which was due to—

Phthisis	in 17
Laceration of the lung . .	in 4
Tracheotomy	in 3
Pulmonary thrombosis . .	in 1
Necrosed pulmonary tissue .	in 2
Ruptured emphysematous vesicle	in 1

In three of the cases there was a hæmopneumothorax. In the first (90[128]),* a case of phthisis, a large cavity, one and a half inches across, had opened into the pleura, setting up a pneumothorax; hæmorrhage had also taken place from a branch of the pulmonary artery in its wall, so that besides a profuse hæmoptysis, there had been an extravasation of fifteen ounces of blood-clot into the pleura. The other two cases had occurred in children (88[219] and 96[102]) who had been run over; the crush had lacerated the lung without fracturing the ribs. Of six fatal cases in children who had been run over, with laceration of the lung without fracture of the ribs, in two, only blood had extravasated into the pleura; in two, air; and in the remaining two both blood and air.

The list emphasises the well-known predominance of tubercle as the main cause of pneumothorax, and supplies one more instance of a ruptured emphysematous vesicle as a cause of the same.

During this same period there have been sixty-five cases in which at the inspection blood has been found in the pleura; three of these have also been included in the list of cases of pneumothorax. The cases of hæmothorax may be classified as follows:

* The figure 90 refers to the year 1890, and [128] to the number of the inspection.

Traumatism.		
Fractured ribs and lacerated lung .	18	
Fractured ribs without laceration of the lung	13	
Laceration of the lung without fracture of the ribs	4	
Neither fracture of the ribs nor laceration of the lung	4	
	—	39
Cancer		6
Rupture of aortic aneurysm		9
Tubercle		3
HCl poisoning		1
Purpura		1
Undetermined toxic infection		1
Erosion of pulmonary artery due to gangrene .		1
Decomposition		1
Cardiac failure		2
Causation obscure		2

The term hæmothorax should be kept for those cases in which the fluid in the pleura was not merely blood-stained, but formed the major part at any rate. In many cases the fluid consisted almost entirely of blood, and both clot and fluid were present, the amount of the latter being inconsiderable in proportion to the amount of clot; in others it was noted that the contents of the pleura were fluid, and that the blood had not clotted. In some cases there was also, probably, exudation of serum. It is clear, therefore, both as the result of experiment and from some of these recorded cases, that as in the case which forms the subject of the present paper, the extravasated blood may remain unclotted.

In the cases in which an aortic aneurysm had ruptured into the pleura there was always clot as well as fluid. The largest amount was forty-five ounces of fluid and fifty-eight ounces of clot. In two cases blood was found in the pleura the origin of which could not be determined.

Pneumothorax (28 cases).

A. *Due to phthisis*, 17 cases.

85[86], M., æt. 38. Paracentesis of serous fluid in December, in February of foul pus.

87[71], M., æt. 29.

87[368], M., æt. 25. Pneumothorax for two years, requiring frequent paracentesis; chest drained and nine pints let out.

88[68], M., æt. 42.

89[156], M., æt. 34. Pyopneumothorax.

90[84], M., æt. 31. Pneumothorax, with lymph over the surface of the pleura.

90[128], M., æt. 40. Phthisis, with profuse hæmoptysis. At the inspection, fifteen ounces of recent blood-clot were found in the pleura; there was an aperture in the posterior part of the right lower lobe of the lung half an inch across, communicating with a cavity one and a half inches across full of blood-clot; there was also a pneumothorax.

90[346], M., æt. 34. A pneumothorax opened by an aperture half an inch across into a small caseous cavity at the left base.

91[168], M., æt. 27.

91[230], M., æt. 30.

92[11], M., æt. 26. There was a left pneumothorax, which was drained with a Southey's tube; this was followed by a universal emphysema, which gradually diminished. The chest was incised and drained.

92[42], M., æt. 54.

92[234], M., æt. 45.

92[367], M., æt. 24.

95[119], M., æt. 46.

95[124], M., æt. 18.

96[155], M., æt. 18.

B. *Due to injury,* 4 cases.

When fractured ribs tear the lung and lacerate the pleura a surgical emphysema results more frequently than a pneumothorax, as the pressure in the connective tissue being lower than that in the pleura during expiration, the air in the pleura is driven into the connective tissue.

88[219], M., æt. 10. The chest wall and ribs were uninjured. The right lung in front had a tear two inches long, and a quarter of an inch deep. The edges adhered together, and but little inflammatory change had taken place. The root of the right lung was torn in two places, one tear being anteriorly over the root of the middle and upper lobes one inch long, and the other posteriorly over the root of the upper lobe.

The right lung was collapsed, and the cavity contained eight ounces of fluid blood and serum. The left pleura contained eighty ounces of blood and serum, the source of which

was not obvious. The lower posterior portion was airless and sank in water; the posterior portion of the lower lobe contained less air than the upper. The condition was one of compression rather than collapse. There was a pneumothorax on the right side; the patient died on the same day.

91^{112}, F., æt. 44. Fell twenty feet and fractured the base of her skull. There was surgical emphysema over the right chest with a pneumothorax. The clavicle and the first to the tenth ribs were fractured on that side.

95^{46}, M., æt. 79, was knocked down by a train. The second to the twelfth ribs were fractured on the right side, and the first to the ninth on the left; both lungs were lacerated, and a double pneumothorax developed.

96^{102}, M., æt. 3, run over; the root of the right lung was lacerated, tearing the tissue without lacerating the large vessels. No ribs were fractured; there was an ounce of blood in the right pleura, with a pneumothorax.

C. *Following tracheotomy*, 3 cases.

93^{154}, M., æt. 28. Was admitted for syphilitic laryngitis and tracheitis, for which tracheotomy had to be performed. After some time dyspnœa ensued, which was followed by violent fits of coughing; this was succeeded by a right pneumothorax, which terminated fatally.

The mechanism which appears to produce pneumothorax in these cases appears to be as follows. The tube becomes slightly displaced, or the obstruction is not relieved, which leads to inspiratory dyspnœa. As the air does not pass readily under these circumstances down the tube, it is sucked down with inspiration into the layers of the median connective tissue, which had been divided at the time of the operation. With expiration the air is driven along the connective tissue, and much of it fails to escape. Ultimately the increasing tension leads to a minute perforation in the pleura, which now allows a ready means of exit for the imprisoned air. A valvular mechanism is thus formed, so that the air is sucked into the pleura with each respiration, and soon the lung becomes completely compressed. The development after tracheotomy of pneumothorax is rare; I remember a previous case, and Dr. Moxon used to refer to this causation.

Dr. Champneys refers to a case in his paper in the *Med.-Chir. Trans.* upon emphysema. Osler also refers to this as a not uncommon cause; but Ziemssen, Quain, Barker, and others fail to refer to its occurrence.

95[229], M., æt. 12. Admitted with urgent dyspnœa, which at first was thought to be due to asthma, but on account of the extreme deficiency in the entry of air it was decided that tracheotomy should be performed, to relieve what was possibly a laryngeal obstruction. This was done on July 1; on the 4th a left pneumothorax developed, and on the 7th there was a fresh attack of dyspnœa, which culminated in the patient's death.

At the inspection both lungs were found to be studded with a saccular form of bronchiectasis; there was no laryngeal obstruction. A double pneumothorax had resulted from the tracheotomy.

97[44], M., æt. 3. Admitted with laryngeal obstruction due to diphtheria, for which tracheotomy was performed. Surgical emphysema of the anterior mediastinum developed, which was succeeded by a right pneumothorax.

D. *Secondary to pulmonary thrombosis,* 1 case.

86[239], M., æt. 38. Wide-spread ante-mortem thrombosis in the heart, lungs, and veins. Right pyopneumothorax.

E. *Necrosis of pulmonary tissue,* 2 cases.

97[150], M., æt. 20. Fifty ounces of foul sero-pus were drawn off from the left pleura.

At the inspection a large gangrenous cavity was found at the base of the lung, three inches by four in diameter, with sloughing walls.

98[181], M., æt. 40. Admitted with cellulitis of his hand. An abscess developed in his lung. A pyopneumothorax developed, and half a pint of pus was let out. There was no hæmorrhage.

F. *Ruptured emphysematous vesicle,* 1 case.

93[113], M., æt. 54. He was subject to chronic bronchitis, and the day before his death was seized with a sudden attack of dyspnœa. At the inspection numerous bullæ were present on the surface of both lungs, and there was extensive emphysema. The right lung was compressed by a large pneumothorax; there was recent lymph over the surface. The lung was carefully distended under water through the bronchi, in order to determine the seat of rupture, but none could be found. There was little doubt that one of the vesicles had given way.

HÆMOTHORAX (65 cases).

A. *Due to traumatism*, 39 cases.

1. *Fracture of ribs, with laceration of the lung*, 18 *cases.*

85[203], F., æt. 32. Had jumped off London Bridge, fracturing her ribs and lacerating her lung. The right pleura contained five ounces, and the left two ounces of blood, which had clotted.

87[269], M., æt. 3. Run over. Two drachms of blood in the pleura.

88[398], M., æt. 40. The first to the seventh ribs were fractured, tearing the lung and extravasating blood into the pleura.

88[403], M., æt. 6. Run over, tearing the lung at its right root, and leading to extravasation of blood into the pleura. The right clavicle and the first to the ninth ribs were fractured, but neither lung nor pleura was lacerated by the fractured ribs.

89[164], M., æt. 53. Run over, fracturing the first to the tenth ribs on the left side. There were over ten ounces of blood in the left pleura. The lung was punctured in four places, one of the lacerations being one and a half inches deep and one and a half wide.

89[167], M., æt. 19. Fell fifty feet. There was a small superficial tear on the posterior surface of the left lower lobe, due to a fractured twelfth rib. There was blood in the pleura.

89[202], M., æt. 35. Crushed. The ribs on the right side were fractured, tearing both the pleura and the lung; there was blood between the pleura and the chest wall, and two ounces of blood in the pleural cavity.

90[212], M., æt. 26. Run over, fracturing the first and second ribs on the left side, and the sternum; the upper lobe of the lung was lacerated. There were four ounces of blood in the pleura; the lung, especially at the base, was engorged with blood and deficient in air; there were subpleural ecchymoses, and blood was extravasated into the lung tissue.

90[226], M., æt. 71. Fell four feet, fracturing the first to the seventh ribs on the right side; he lived twelve days. The parietal pleura was lacerated; there was a pint of blood-stained fluid in the right pleura. There was lymph on the surface of both lungs, with a condition of pneumonia, which was becoming gangrenous.

90[266], M., æt. 40. Received a blow from a lever, which fractured the fifth and the eighth to the eleventh ribs on the

left side. He lived seven days. The lung and pleura were lacerated, and there was over a pint of bloody fluid in the pleura, with lymph over its surface.

90[380], M., æt. 37. Run over by a train. All the ribs on the right side were fractured; the lung was lacerated; there were two pints of blood, which had not coagulated, in the pleura.

91[165], M., æt. 24. Railway smash. The ribs on the right side were fractured, the lung lacerated, and there were seven ounces of blood in the pleura.

92[147], F., æt. 46. Run over, fracturing all the ribs on the left side. The lung was lacerated and compressed, and there were thirty ounces of blood in the pleura.

93[189], M., æt. 49. Crushed, all the ribs on the right side with the exception of the last were fractured; the lung was lacerated; there was half a pint of blood in the pleura.

95[257], M., æt. 37. Run over. The fifth to the ninth ribs on the left side were fractured; the lung was lacerated; the left pleura contained twenty-two ounces of blood.

95[288], M., æt. 50. Crushed in a barge. The first to the tenth ribs on the left side were broken; the lung was punctured, and the pleura contained seven ounces of blood. The laceration was filled up with an indurated hæmorrhage.

95[447], M., æt. 23. Fell twenty feet, fracturing the third to the seventh ribs on the right side, lacerating the lung; the pleura contained blood, also a pneumothorax.

2. *Fracture of the ribs, but no laceration of the lung,* 13 cases.

88[85], M., æt. 56. Railway accident. Ribs fractured, subcutaneous emphysema. Four and a half pints of darkly stained serum, with altered blood-pigment, in the pleura. Here there had probably been a hæmopneumothorax, but the air had been expelled into the subcutaneous tissue.

88[328], M., æt. 40. Fell down a ship's hold, fracturing the last six ribs on the right side. The pleura contained fifty ounces of blood.

89[105], M., æt. 44. Fractured all his ribs on the right side, and the third to the seventh on the left. There were subpleural ecchymoses. The right pleura contained twelve ounces of blood, and the left two.

89[374], M., æt. 60. Fell off a van, fracturing the third, fourth, and fifth ribs on the right side; the pleura contained four ounces of blood-stained fluid.

90[87], W., æt. 29. Fell off London Bridge, striking a but-

tress. Several ribs were fractured on each side; there was a little blood in the left pleura.

91[254], M., æt. 69. Crushed by a bale. The second to the twelfth ribs on the left side were fractured. There were six ounces of bloody fluid in the left pleura. There was healed phthisis at both apices.

91[369], M., æt. 50. Twenty ribs fractured. There were twenty ounces of bloody fluid on the right side, eight on the left.

92[226], M., æt. 56. Dragged along under the wheel of an omnibus. He lived seven days. The third to the eleventh ribs fractured. Three ounces of bloody fluid with lymph in pleura.

92[389], M., æt. 3. Run over. The first to the tenth ribs on the right side fractured. The lung was not torn; the pleura contained a pint of blood.

93[231], M., æt. 8. Run over. The fifth to the seventh ribs on the left side fractured; the pleura was not torn. The left chest was full of blood, and there was blood in the posterior mediastinum.

93[472], M., æt. 64. Crushed. The first to the tenth ribs on the right side were fractured. There were fifteen ounces of blood in the pleura.

92[359], M., æt. 40. Fell two stories, fracturing the sixth to the tenth ribs on the left side. Blood in the pleura.

97[243], W., æt. 5. Run over. Six ribs were fractured on the right side. There was no laceration of the lung; there was surgical emphysema beneath the right pleura on the right side. The right pleura contained five, and the left four ounces of blood. This probably commenced as a hæmopneumothorax.

3. *Laceration of lungs, without fracture of ribs,* 4 cases.

87[414], M., æt. 7. Run over. Right lung lacerated; six ounces of bloody fluid in the pleura.

95[125], F., æt. 2. Run over. The right lower lobe was extensively lacerated, with a large triradiate rupture. Two ounces of blood in the pleura. No ribs were fractured.

88[219]. Hæmopneumothorax (see p. 105).

96[102]. Hæmopneumothorax (see p. 105).

4. *Neither fracture of ribs nor laceration of the lung,* 4 cases.

87[49], M., æt. 49. Chest crushed. Three quarters of a pint of blood in the left chest, with some extravasation of blood

beneath the parietal pleura. No fracture or ulceration could be found.

90[477], M., æt. 23. Brought in dead, a load of bricks having fallen on his head. The skull was fractured; no ribs were broken. There were five ounces of bloody fluid in the right pleura. There was ecchymosis in the posterior mediastinum and beneath the pleuræ of both lungs; the lungs were spotted with aspirated blood, but had not been lacerated.

91[255], M., æt. 24. He had been run over. There was a hernia of the stomach up into the thorax, through the diaphragm, which had been ruptured. There was half a pint of blood in the left pleura.

95[379], M., æt. 9. Had been run over. There was a quantity of bloody fluid in both pleuræ. The ribs were not fractured, nor was the lung lacerated. There was a subpleural hæmorrhage at the right base near the aorta. The stomach, kidneys, spleen, and diaphragm were also ruptured.

B. *Cancer,* 6 cases.

85[284], M., æt. 22. Cancer of the lung. A blood-stained fluid with a large amount of clot was found in the pleura.

88[10], F., æt. 61. Primary cancer of the pleura. There were twenty-six ounces of blood-stained serum in the pleura.

89[172], F., æt. 54. Cancer of the breast and pleura. The right pleura contained thirty ounces, and the left twenty-four ounces of blood-stained serum.

90[110], M., æt. 36. Sarcoma of the omentum and pleura. In the right pleura there were five and a half pints of blood-stained serum. At the apex there were four small fibroid nodules, the remains of old tuberculous disease, but there was no recent disease.

91[266], M., æt. 23. Malignant growth in the mediastinum. One pint of fluid, deeply blood-stained, in the right pleura.

93[73], M., æt. 35. Sarcoma of the mediastinum. Twenty-four ounces of bloody fluid in the left pleura.

C. *Aortic aneurysm,* 9 cases.

85[213], F., æt. 40. An aortic aneurysm had ruptured into the pleura.

86[75], F., æt. 41. A dissecting aneurysm had apparently opened into the mediastinum. The left pleura contained twenty-five ounces of blood. Probably there was a minute aperture in the wall of the aneurysm, but it was not discovered.

86[196], F., æt. 40. An aneurysm had opened into the right pleura, which contained three pints of clot and serum.

89^{42}, M., æt. 36. An aneurysm of the aorta had ruptured into the left pleura, which contained eighty-five ounces of blood-clot and serum.

90^{196}, F., æt. 51. An aortic aneurysm had ruptured into the right pleura, which contained more than three pints of blood-clot and serum.

95^{358}, M., æt. 29. An aneurysm had ruptured into the left pleura, which contained fifty-five ounces of clot and serum.

95^{461}, M., æt. 56. An aneurysm had ruptured into the right pleura, which contained fourteen ounces of clot and a quantity of fluid blood.

96^{209}, M., æt. 46. An aneurysm had ruptured into the left pleura, which contained sixty-eight ounces of blood and clot.

96^{442}, M., æt. 31. An aneurysm had ruptured into the left pleura, which contained forty-five ounces of bloody fluid, and fifty-three ounces of clot. The right lung was over-distended, it weighed twenty-one ounces; while the left weighed eight ounces, *i. e.* twelve ounces of blood had been squeezed out of the left lung.

D. *Tubercle*, 3 cases.

88^{361}, F., æt. 30. At the inspection there was a caseous broncho-pneumonia with tuberculous pleurisy. There were fourteen ounces of blood-stained fluid in the pleura.

90^{41}, M., æt. 48. Cirrhosis of the liver with ascites. Twenty-two ounces of blood-stained fluid were drained off from the right pleura. At the inspection grey tubercles were seen scattered through the left lung and on the pleura. The right pleura was enormously thickened, and in the midst of the thickened membranes there were fourteen ounces of blood-stained fluid; the lung was compressed to the size of a fist.

90^{128}. Hæmopneumothorax (see p. 104).

MISCELLANEOUS (8 cases).

Due to HCl *poisoning.*

85^{278}, M. HCl poisoning. There was a subdiaphragmatic abscess. The pleura contained some bloody purulent fluid.

Purpura.

87^{416}, M., æt. 27. Purpura. The pleura contained eighteen ounces of blood-stained serum.

Probably due to a toxic infection, the nature of which was not ascertained.

88^{423}, M., æt. 30, who died with a subdural hæmorrhage. The right pleura contained twenty-six ounces of liquid blood, and the left five ounces. The pleuræ were normal; the lungs were intensely congested with dark blood.

Gangrene. Ulceration of pulmonary artery.

91^{119}, M., æt. 45. Pneumonia of the right lung followed by gangrene, ulcerating into the pulmonary artery, the aperture being the size of a crow quill. Fifty ounces of clotted blood were found in the right pleura.

Decomposition.

88^{319}, F., æt. 39, who died with a strangulated omental hernia. The body had decomposed greatly, the viscera were emphysematous; there were three ounces of blood-stained fluid in the pleura.

Cardiac failure.

88^{372}, M., æt. 19, who died with malignant endocarditis of the mitral, aortic, and tricuspid valves. There were eight ounces of blood-stained fluid in each pleura. There were no infarcts; the bases of the lungs were splenised and solid.

87^{156}, M., æt. 49. The patient died of cardiac failure with an hypertrophied heart. There was a pulmonary apoplexy. The pleura contained six ounces of blood-stained fluid.

Causation obscure.

96^{30}, M., æt. 1. Tracheotomy was performed for diphtheritic laryngeal obstruction, and artificial respiration was performed. The left pleura was normal, but the right contained two and a half ounces of blood and clot, the causation of which effusion remains obscure.

88^{232}, M. æt. 35, brought in dead, having died suddenly. The right pleura was firmly adherent behind; the adhesions were black from soaking in blood. There was half a pint of fluid blood in the front of the upper portion of the right lung, in the pleura, and nine at the base; there was no clot, and no source could be found for the blood. The aorta, the vena cava, and their main branches were healthy. The sternum had been removed when I saw the body. There was no evidence that the condition of the blood was abnormal,

and the causation of the hæmorrhage remains an open question. The absence of any clot is curious. It is just possible that the hæmorrhage took place after the thorax was opened, but I came to the conclusion at the time that the hæmorrhage was *ante-mortem*. There were a few adhesions in the left pleura. There was some frothy blood in the bronchi, with some mottling in the lungs posteriorly; there was no clot in the bronchi, and the amount of blood was small. The lungs were free from disease, except for a small patch consisting of nodules of caseous broncho-pneumonia surrounded by fibroid tissue. The nodules were a tenth of an inch across, and the whole mass measured an inch; it was situated at the apex. There was no cavitation, and no hæmoptysis near the spot. The rest of the lung was normal; the base was deficient in air. The heart weighed sixteen ounces, the left ventricle was greatly hypertrophied with slight dilatation of its cavity. The aortic valves were thickened but competent; the coronary arteries were rigid and thickened.

Unfortunately, the right bronchus was torn in pulling the lungs out afterwards, but there was no evidence of any clot near it, nor any evidence of hæmorrhage having occurred near it. I do not think this was the source of the blood, although it is possible that the bronchus may have been torn during life and the laceration increased after death. The kidneys weighed ten and a half ounces, their surface was granular; the cortex was congested, and was below the average in size.

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XVI.—*A case of Perforating Duodenal Ulcer; operation; recovery.* By J. J. Perkins, M.B., and C. S. Wallace. *Read February* 19, 1900.

A. W. A., male, æt. 52, carman, a strong healthy man, with a good family and previous history, was admitted to St. Thomas's Hospital on August 12th, 1899.

For three days previously he had suffered from some slight pain in the epigastrium, which was not aggravated by food or movement.

On the day of admission, at 6 P.M., after partaking of a meal consisting of a mutton-chop, tea, and bread, he was seized with a violent pain in the epigastrium, so severe that it doubled him up, and rendered the erect position impossible. A doctor sent for an hour later prescribed some medicine, but this failing to relieve, he ordered his removal to the hospital at midnight.

No vomiting.

When seen the abdomen was slightly distended in the upper third, tense, rigid, and moving but slightly on respiration. There was no abnormal dulness, but there was a resonant note over the liver area in the nipple line, the liver dulness being completely abolished.

Pulse 85, temperature 99°, respirations 26. The facial expression was anxious, and the pain in the abdomen evidently very great, occurring in paroxysms.

From the age of the patient, the history, and the absence of liver dulness, it appeared most probable that a duodenal ulcer had perforated.

Operation.—At 4.30 a.m. (August 13), ten and a half hours after the onset, the abdomen was opened through the right rectus, just to one side of the middle line. Immediately below the incision was some greenish viscid fluid, mixed with some white flakes of lymph or curdled milk. The intestines here showed some points of injection, but there were no very evident signs of peritonitis. The stomach was rather distended, and full of fluid contents. The duodenum was then sought for, and after some little search, in which the incision was enlarged to the umbilicus, a perforation was found with a diameter of a third of an inch, situated almost at the

junction of the first and second portions of the duodenum on the anterior surface. The edges were clean cut, and showed no sign of induration. The perforation was closed with two rows of sutures. Considerable difficulty was experienced in doing this, as the ulcer seemed very deep, and the abdominal wall would not relax, though chloroform was used.

The abdomen was then cleaned with evisceration and flushing (with sterilised water), after which the wound was closed with silkworm gut sutures, including the whole wall, and by a row of buried sutures to the anterior rectal sheath. A gauze wick was left in, passing down to the seat of perforation. No other drainage.

Convalescence was uneventful; the plug was removed on the second day, and the bowels were opened by enema on the third day.

On August 14 the abdomen was noted to move well, and to be nowhere rigid. For the first ten days only soda water was allowed by the mouth, milk being first given on August 24, and rectal feeding discontinued on August 27, on which date jelly, arrowroot, and custard were first added to the milk diet.

The case read is one of five cases of perforation of a duodenal ulcer admitted to St. Thomas's Hospital within the last three years.

Other instances of operation and recovery have been reported by Mr. Dean and Mr. Dunn, and one of abscess due presumably to duodenal perforation by Mr. Howard Barrett. Messrs. Lockwood and Sheild previously did much to draw attention to the condition, and to the possibility of operative success.

Drs. Perry and Shaw in their article on the Diseases of the Duodenum (*Guy's Hospital Reports*, 1893) have collected all the cases of duodenal perforation published in this country, making with those mentioned 64 cases; ulcers following burns and other septic conditions are excluded.

The site of the perforating ulcer of the duodenum has attracted attention. In 37 of these 64 cases exact details of the site are given; in 29 it was anterior; in 8 posterior or partly posterior. This is in marked contrast with the site of other ulcers of the duodenum, where perforation did not occur, but hæmorrhage or some intercurrent disease was the cause of death.

Of 32 cases (Perry and Shaw) of death from hæmorrhage, in twelve the ulcer was posterior; in 18 its base was formed

of the pancreas; in 1 both anterior and posterior existed; in 1 the site was not given.

Not only, however, is the condition very suitable for surgical interference, but these cases provide a strong argument for immediate operation. Though life was prolonged in one instance for seven days, the term was usually much shorter, and in 13 of the cases (not operated on) death occurred within twenty-four hours, the average duration of life being seventeen hours. In one case, too, which survived thirteen hours, "universal" peritonitis was found at the post-mortem examination, while in another surviving twenty-four hours the peritonitis is described as "intense."

The rigid contracted condition of the abdominal wall present in our case is noted in several of the reports, and with the paroxysmal pain, due to peristalsis, led to the diagnosis of colic in more than one instance. The loss of liver dulness in and internal to the nipple line combined with such a condition of the abdomen is surely strong evidence of perforation. As regards the help in diagnosis to be derived from the previous history of the patient, in 15 instances there was a history strongly pointing to the duodenum, in 14 the history was more or less vague and weak, while in 16 the health up to the moment of rupture is noted as good or excellent. This absence of symptoms the perforating shares with the other forms of duodenal ulcer. Perry and Shaw, out of 151 cases, of which the records were fairly complete, found 91 without any record of noticeable symptoms. They further show that the duodenal cannot be diagnosed from the gastric ulcer, either by the site of pain when felt, or the interval between food and the onset of pain. Vomiting after perforation, which is held by some to be distinctive of the duodenum as contrasted with the stomach, is noticeably rare in these 64 cases.

The records of these 64 cases are not sufficiently full to decide whether the site of pain and tenderness after perforation is at all a constant guide. Instances are given where the reference is misleading, *e. g.* the right iliac fossa, to be explained, as Mr. Sheild suggests, by peritonitis starting at a distant point from extravasation of contents.

The history, then, may be of help in a considerable proportion of cases, the site of pain and tenderness will probably aid as a rule, and after opening the abdomen the non-fæcal character of the extravasation.

Gastric and duodenal ulcer are indistinguishable except by age and sex of patient.

Result.— In only three cases (occurring in Perry and Shaw's list) did the disease run a chronic course, with localised abscess formation, one patient surviving six months, one four years, and one five weeks. In the first multiple intra-peritoneal abscesses formed; in the second the abscess opened in the back; in the third the abscess lay between the liver, the anterior abdominal wall, and surrounding coils of intestine. Localised abscess formation in association with general peritonitis is reported in another case, a large abscess lying between the liver, the stomach and duodenum, elsewhere shut in by adhesions between the intestines. Lockwood and Sheild have reported cases of abscesses in a similar situation, also in association with peritonitis. In one of the cases occurring at our own hospital there was an abscess running up over the right lobe of the liver, reaching downwards to the anterior superior iliac spine shut in by the great omentum.

In all the other cases diffuse peritonitis resulted, which in the earlier cases was usually found to be general after death. To learn its mode of spread one must take only the cases of later years, where operation has been undertaken at an early date.

Such peritonitis may be general, as in Mr. Dean's case (operation twenty-four hours after perforation). It may, however, take a different course, the escaped contents of the bowel tending, as in perforated gastric ulcer, to pass down over the great omentum, and gain the pelvis without the great mass of the intestines being infected. This condition was present in one of our cases at St. Thomas's, where much lymph was present in the intestines about the parts involved, and also much fluid in the pelvis, but the intestines only showed some small points of injection (operation forty-eight hours after perforation). In the case of abscess referred to above, the abscess lay on the anterior surface of the great omentum in the right half of the abdomen, and was prevented from extending downwards and to the left only by the close apposition of the omentum to the anterior abdominal wall, and that without adhesion. A similar localisation and restriction of peritonitis is recorded in one of Perry and Shaw's cases.

In one of Mr. Lockwood's cases it is said the abdomen might be divided into three zones—lowest of all a zone of septic peritonitis, the pelvis containing at the operation Oij of

pus, a middle wide zone almost free from peritonitis, and lastly, beneath the liver, a zone of septic peritonitis spreading from a large abscess.

If, however, the cases are not immediately fatal, or are not relieved, the peritonitis spreads down between the liver and the meso-colon. Arrived at the edge of the liver it ascends over the upper surface of that organ, mostly to the right of the falciform ligament, but sometimes also to the left, and may then reach the spleen. At the same time it has probably followed another route, and passed out along the duodenum to the anterior surface of the right kidney. This spot when the patient is lying on the back is really one of the lowest in the abdomen, and forms a sink where much pus can collect. It next ascends over the surface of the liver, and here meets the peritonitis rising from the anterior edge of the liver.

This spread upwards over the liver was seen in the case of abscess already referred to, and in another case (operation forty-eight hours after perforation) the upper surface of the liver was coated with fibrin, and a small quantity of fluid was here liberated.

Technique.—The whole abdomen should be cleansed by irrigation, combined with evisceration of its contents, special attention being paid to the sites above mentioned (pelvis, upper aspect of liver, etc.). A gauze drain should be inserted down to the site of the ulcer; general drainage is not recommended.

The collapse, which often forms so marked a feature in duodenal perforation, and is intense, was successfully met in one of the cases at our hospital by saline infusion combined with hypodermic injections of strychnine and brandy, enabling operation to be carried out on the following day.

XVII.—*A case of Stricture of the Small Intestine following Strangulated Hernia.* By Thomas H. Kellock. *Read February* 9, 1900.

J. B., æt. 16, indoor porter, admitted to Middlesex Hospital September 3, 1899.

Family history.—Father died of cancer of the bowels. Mother and five brothers and sisters living. One brother dead.

Previous history.—Had measles when three years old, and was laid up one week with influenza in 1892. In the summer of 1897 was operated upon at the West London Hospital for hydrocele in connection with left partially descended testicle. After the operation a hernia appeared occasionally in the left inguinal region; he had no difficulty in reducing it when it came down, but he did not wear a truss.

On the morning of the day of admission to the hospital patient strained himself, the hernia came down, and he could not reduce it; he experienced a good deal of pain in it and was very sick. On admission in the middle of the following night he was very collapsed, and was vomiting a good deal; there was a swelling the size of an orange in the left inguinal region, which did not extend into the scrotum; it was irreducible, tender, and resonant on percussion, except at its lowest part.

The right testicle was in the scrotum, but the left could not be felt. An anæsthetic having been given an incision was made over the swelling; on opening the sac it was found to contain no fluid, but was occupied by a small piece of discoloured omentum and about 4½ inches of small intestine, which was almost black but the surface glistening.

After the stricture at the neck of the sac had been divided, the piece of omentum ligatured above the discoloured part and removed, a little more of the bowel was drawn down; a marked constriction was found at both ends; but after a short time the colour of the bowel seemed to improve, and it was therefore returned to the abdomen. Lying internal to the sac the testicle was found; it was small and ill-developed. There was a good deal of fluid in the tunica vaginalis, which latter did not communicate with the abdominal cavity or with the sac of the hernia. The spermatic cord was ligatured, the

testicle removed, the inguinal canal closed by two silk sutures, and the skin wound united with horsehair.

During the following twenty-four days there was nothing very much to note; the youth was drowsy and apathetic, but that appeared to be his normal condition. The wound healed by first intention, and his temperature and pulse continued practically normal. There was a little difficulty in getting the bowels to act, but they responded to enemata and mild aperients. He complained of feeling sick several times, and vomited on two occasions. On September 16 (thirteen days after the operation) the abdomen was a little distended and slightly tender, but this improved when the bowels had acted again.

On the twenty-fourth day after the operation he seemed to be progressing favourably and was taking a little solid food, but on the following morning he complained of pain in the abdomen, vomited several times, and had a rigor which lasted fifteen minutes, the temperature rising to 102·2°. The abdomen was found to be tense, distended, and very resonant on percussion. Peristaltic action of the intestines could be seen and felt through the abdominal wall. The tongue was brown and furred, and the patient very drowsy. An enema was given with a very good result, the motion being formed. On the following day the temperature was normal again, and all the symptoms had subsided. On October 4 he was sick after eating some biscuits given him by his friends, but with that exception he progressed favourably and was sent to a convalescent home on October 11; a note on that day states "the bowels are acting naturally, he has no pain, and there is no distension of the abdomen." During his stay of three weeks at the convalescent home he suffered at times from pain in the epigastrium, and was sick on several occasions, vomiting undigested food soon after his meals. The bowels were somewhat constipated, but were relieved by aperients. After leaving the convalescent home he returned to his home, and was seen at the hospital occasionally as an out-patient. He still complained of pain in the abdomen and occasional sickness, but they were both relieved by saline aperients. It was thought at this time that the trouble was due to adhesions about the part of the bowel that had been strangulated, but it was not thought advisable to subject him to any operation.

On November 26, whilst at home, he had a bad headache, felt very hot, and had severe pain in the epigastrium. He was very sick during the night, bringing up whitish fluid; the

bowels were open two or three times during the night, the motions being green in colour and quite liquid; the following day he felt much better, but the diarrhœa continued.

He was readmitted to the hospital on November 28, under the care of Dr. Fowler, diarrhœa, vomiting, and epigastric pain being the symptoms complained of. The abdomen was a little distended and very resonant on percussion, but not tender, and moved well with respiration. The tongue was white and furred, temperature 98·2°, pulse 78. His condition improved somewhat during the succeeding day or two, but on the morning of December 3 he was much worse. The vomiting had increased and the diarrhœa had become rather profuse; the abdomen was more distended and peristalsis visible. The stomach was washed out, but without much result. A hypodermic injection of morphia was given, and a hot compress put on the abdomen. In the afternoon his condition had improved slightly, and then the abdomen was opened in the middle line below the umbilicus. Distended hypertrophied small intestine presented immediately; there was no peritonitis, and the cause of the obstruction was easily and quickly found, for lying in the right iliac fossa was a loop of small bowel to which a tongue-shaped piece of omentum containing large vessels was closely adherent. The distension of the intestine terminated abruptly at this spot, that below the loop being of normal size. In the loop of intestine a tight hard stricture could be felt completely surrounding it. As it was partly obscured by the adherent omentum this was ligatured in three pieces and divided. The intestine was then examined for some distance in both directions, and no other abnormality being found a lateral anastomosis of the intestine above and below the stricture was made by Halstead's method and the abdomen closed.

The patient was a good deal collapsed after the operation, but revived somewhat after repeated small enemata of hot water with a little brandy and a hypodermic injection of strychnine. On the third day the bowels acted twice, the motion being quite liquid; he also passed a quantity of flatus. In the afternoon of that day the abdomen was found to be consideraby distended, tender all over, and not moving with respiration; the pulse was very rapid and weak, and the general condition very bad. On the supposition that there had been a leak at the site of the anastomosis the abdominal wound was reopened without anæsthetic. The peritoneum was found to be inflamed, and there was a quantity of lymph adherent to

the bowel at the seat of the operation. The bowel above the anastomosis being much less distended than at the time of the operation, and the patient's condition preventing anything further being done, a drainage-tube was inserted reaching down to the position of the anastomosis, and the abdominal wound closed round it. Death occurred about fifteen hours afterwards.

I am indebted to Dr. Campbell Thomson for the following account of the condition found at the *post-mortem* examination.

About three feet above the cæcum a lateral anastomosis had been made between two adjacent parts of a coil of small intestine. The sutures uniting the two coils together were quite firm and watertight, and the aperture leading from the one coil to the other was quite freely open, and could have offered no resistance to the passage of fæces. The loop of intestine, which the anastomosis was designed to put out of action, was of a dark purple colour and quite gangrenous. At one point there was a small perforation through which the liquid contents of the bowel freely passed into the peritoneal cavity. Near the end of this loop there was a small mass of omentum which was firmly adherent to it, and the mesentery attached to this portion of the bowel was rather hard and puckered. The gangrenous portion of intestine was lying in the right iliac fossa. There was a large quantity of pus and fæculent fluid in the peritoneal cavity, and a condition of general peritonitis was present. On opening the gangrenous portion of the bowel an annular stricture was found at the apex of the loop almost completely obstructing the lumen, and involving about half an inch in length of the bowel.

Stricture of the small intestine is generally classed among the possible sequelæ of strangulated hernia, but the rarity of its occurrence, at any rate to the degree of calling for operative measures for its relief, made me think it worth while to report this fairly typical case.

The association of an imperfectly descended testis with inguinal hernia is of course a common one, but it is, I believe, generally taught that the herniæ in these cases are commonly of the congenital or funicular varieties. This, however, was not so in this case; the hernia appeared to be an acquired one, the processus vaginalis being occluded at its upper part, leaving the tunica vaginalis in its normal relation to the testis, but these relations may have been somewhat altered by the

previous operation for hydrocele in connection with the imperfectly descended testis; the latter had recurred to some extent, but may of course have been increased by the patient's effort to reduce the hernia.

The condition of the bowel at the first operation was such as to make one doubtful whether to return it or adopt one of the alternatives—resection or the formation of an artificial anus. The facts, that although very discoloured it had not lost its shining appearance, that after relieving the stricture and drawing down a little uninjured gut the colour in the portion that had been strangulated improved somewhat, and the patient's age being that at which the powers of repair should be at their best, made me decide on returning it. It should be mentioned that at the time of the operation no marked constriction or abnormality was noticed in the mesentery of the bowel that had been strangulated. It would appear that the stricture of the intestine began to make itself felt about a fortnight after the operation, for the occasional pains in the abdomen, nausea, and at times actual vomiting, which began to occur at that time, must be attributed to it, and from then onwards, as the notes show, these symptoms were seldom absent for any length of time and became gradually more marked. The alternating constipation and diarrhœa characteristic of stricture, even in the small intestine when the contents are generally liquid, were pronounced; towards the termination of the case peristalsis was a troublesome feature, and the diarrhœa was continuous.

The interest in the condition found at the second operation centres itself in the tongue-shaped piece of omentum which was adherent to the bowel immediately distal to the stricture; it was noticed at the time that it was rather more vascular than usual, but the importance of this fact did not strike one then. What had occurred was evident subsequently; the mesentery attached to the strangulated bowel had been itself so constricted that the blood-vessels in it had been more or less occluded, and when the bowel was returned to the abdomen it had received its blood supply, not through its own mesentery, but from this piece of omentum which had become adherent to it. Fortunately it may be, or possibly if this had not occurred the circulation through the mesentery might have been re-established. The cutting off of this blood supply when the omentum was ligatured and divided, resulted in the death of a small piece of the intestine, the subsequent gangrene, leakage, and fatal peritonitis. Whether the adherent

piece of omentum was the same piece as I had originally ligatured in the first operation I have been unable to determine, but I should think it highly probable.

It was feared that leakage at the anastomosis was the cause of the peritonitis, and the satisfaction of finding this intact and in good working order was a good deal damped by finding the actual cause of death one that might possibly have been avoided; but this unfortunate result in a case which otherwise promised so well cannot fail to be a valuable and instructive lesson.

XVIII.—*On the Prevention of Valvular Disease in Acute Rheumatism.* By Richard Caton, M.D. *Read March* 9, 1900.

NO physician who has had long experience in hospital or private practice can escape being impressed by the number and the gravity of the cases of organic heart disease which come to him hoping for the cure which is out of his power to accomplish, or for the relief which he knows at the best can only be temporary. Amid these experiences many of us have asked ourselves the question, if we can never hope to cure, is it impossible to prevent such sad disabling of the circulatory mechanism? Must we in particular always remain mere passive spectators of this maiming of the heart in acute rheumatism? At the present time by the great majority of our profession all preventive measures are regarded as futile, and no energetic or determined effort is made to combat the endocardial mischief which, at its onset, the practitioner recognises with so much concern and with so little hope.

This I confess was my own attitude during the first twelve years of professional life, but nineteen years ago, after inquiring into similar attempts by others, I began a series of experiments in the way of prevention, which for the first four years were almost entirely without any good result. During the last fifteen years, however, the issue has been different, and as one began to feel the way to practical methods, and learned to avoid mistakes, has become highly encouraging. I wish briefly to state the method adopted and the results, avoiding all details not absolutely needful.

I may state that my cases of febrile rheumatism have amounted to nearly 500, that the general treatment adopted was the usual one of appropriate doses of salicylates, combined sometimes with alkalies, always preceded by a cholagogue. The patient is invariably clothed from head to foot in flannel, and kept strictly and always in bed for some weeks, and on a simple diet, red meat being excluded. In case of pain lingering in any joint it is dispelled by the use of small blisters locally.

The absolute rest and the avoidance of chills during and after the pyrexial period lessen, I think, the liability to cardiac complication. My proportion of cases of threatened cardiac trouble is under 20 per cent. of the total number.

Before attempting to define endocarditis or to state the physical signs relied on to indicate its presence, it will be well to say that the brief bruits or murmurs, lasting perhaps a day only, which we sometimes hear, and the causes of which personally I do not understand, are excluded from consideration; so also are all hæmic bruits. Murmurs arising from dilatation are sometimes more difficult to discriminate from those of valvulitis, their main characteristic being increased cardiac dulness; where emphysema exists the diagnosis is not always easy. They are in my experience not particularly common, but I have endeavoured always to keep them in mind; sometimes valvulitis co-exists with dilatation. It is, however, possible that some few cases in my list may have been mistaken by me for valvulitis when the cause of the bruit really was dilatation. I must say, however, that in no case was careful percussion of the heart omitted.

The signs chiefly relied upon have been the following:—during, or immediately subsequent to the attack of febrile rheumatism, the cardiac dulness being normal, a change has become manifest in the character of the first sound at the apex, an indistinctness called by the French *assourdissement*, followed by development of bruit, usually systolic at the mitral, commonly heard in the axilla, with subsequent accentuation of the second pulmonary sound, or more rarely by a diastolic or a systolic bruit at the aortic valve.

I have been rather surprised at the comparative rareness of valvulitis of the aortic orifice. When it has occurred it has appeared to be an extension from a previously affected mitral valve.

When the onset of this train of symptoms has been observed, or when the patient on coming under observation has the systolic apex murmur and second pulmonary accentuation already established, and no increased dulness, I always adopt the three following measures:—first, the most absolute rest in bed in strict recumbency for several weeks is enjoined; second, the application of a series of small blisters, each less than the size of a florin, and each followed by a small poultice in the region of the first, second, third, or fourth dorsal intercostal nerves in front. Only one at a time is applied, the object being to keep up a constant but gentle stimulation of those nerves. Thirdly, sodium or potassium iodide in eight or ten grains dose thrice daily (or even in some cases small doses of mercury) are administered.

Now to explain the theory of these measures. The object

of the rest is of course to keep down the velocity and force of the blood current, to slow cardiac action, to minimise the dangerous pressure of the blood wave on the weakened and softened valve cusps. That indication is clear and obvious. The use of the blisters needs more explanation. The therapeutic value of local stimulation in certain morbid states is well known to the surgeon, most of us have at some period seen the effect of copper sulphate, or silver nitrate, or of other local stimulant in promoting the healing of an ulcer and in many other applications.

In the treatment of rheumatic affections of the joints the influence of local stimulation of the skin is very striking. Probably few here present remember what one may term the pre-salicylic period of acute rheumatism, and consequently few may recall the great value of Dr. Herbert Davies' method of treatment by small blisters to the affected joints. Those who have witnessed it know how effective it was. The rapidity with which swelling and pain disappear from any rheumatic joint after the application of two or three small blisters is very striking. It is doubtless due to stimulation of the trophic nerve filaments of the part (transmitted from the sensory nerves of the skin), whereby arterioles are contracted and increased activity of the natural reparative process is promoted. Now the question suggested itself to me, if stimulation of the surface skin will so potently assist recovery in a rheumatic joint, is it not possible that the same beneficent change might also occur in the heart if continued stimulation of its related skin areas were practised? At first sight the greater distance intervening between the thoracic skin and the heart appeared an objection, but the nerve connection is an intimate one, as those of us know who have observed the referred pains of angina and other heart lesions; and just as ten yards or a thousand matter little in conduction along a telegraph wire, so probably the reflection of an afferent stimulus, *viâ* the sensory neuron and the cells of the cord, the sympathetic or of the posterior root ganglion would present no greater physiological difficulty.

At an early stage in the inquiry I had proved by the use of the galvanometer that stimulation of the surface thoracic skin modified the electrical potential in the muscles of organs within the body-cavity, also that similar stimulation caused changes in the calibre of arterioles in those organs. As vasomotor and electrical functions in these organs were influenced by skin stimulation in the frog, it seemed not unreasonable to

assume the same in regard to vaso-motor function in man, and also in the case of trophic function. Thus there appeared to be an antecedent probability that the channel of nerve influence which Dr. Head and others have pointed out existed, was available for therapeutic purposes. Thirdly, as regards the use of the sodium or potassium iodide. This is given in the hope that it may help in the absorption of imperfectly organised inflammatory products in the valve cusps and endocardium. These I believe are removable in the early stage, but not, at any rate in adults, if they once become fully organised.

Now let me briefly recount the results of this system of treatment. I need not say that it would be useless and absurd to apply the method to old cases of heart disease, in which contraction, necrosis, or adhesion of cusps had occurred, with any expectation of cure resulting. It is only to be employed in those in which the endocarditis is thought to be, or known to be, quite recent. During the last fifteen years, though anxious to obtain them, I have only had eighty-five of these cases in my hospital practice.

A summary of the results is given in the paper I have placed in your hands. It contains only cases recorded by the hospital registrars. I have excluded all treated in private, as they would have been recorded by myself alone.

These cases are divided into two groups:

Firstly, those which, on coming under treatment, already had a bruit and other signs of valvular disease, probably, but not certainly, of recent origin. They number fifty-four; of these thirty-six left the hospital with apparently sound hearts, while the remaining eighteen had, I fear, valvular disease.

Secondly, those cases in which the bruit and other signs of valvular disease came on while the case was under my own treatment, or in which its origin had been recently observed by the medical friend who sent me the case. Here we knew with certainty that we were dealing with recent cases. These patients were thirty-one in number; of these, twenty-seven left the hospital with an apparently sound heart, three had valvular disease, and one is now under treatment. This is a measure of success far greater than I ever hoped to attain. For many years case after case in succession has terminated favourably, so that we have learned to look for success with some confidence, provided the treatment is strictly observed and for a sufficiently long time. And but rarely is one disappointed, in so far as early cases are concerned. So common is

complete disappearance of all signs of cardiac trouble in those in whom its onset had been distinctly marked, that I should feel I was acting wrongly if these measures were in a single case neglected. One important adverse circumstance should be noted, viz. that if, in a case in which under treatment the endocarditis has disappeared, acute rheumatism recurs within a short period, the heart symptoms almost always recur also; under the same treatment they have sometimes again disappeared, but in some cases they persist and lay the foundation of permanent organic mischief.

One important question remains, What was the subsequent history of these sixty-one cases which I have asserted left hospital with a sound heart?

As far as possible I have kept them in view for months or years after, but in the majority of cases they are eventually lost sight of, and in some instances this happens at an early date. In many, however, I have had the satisfaction of examining the patient years after, and of finding the heart sound, and the patient strong and active. The only cases I am acquainted with in which mischief occurred are those in the printed list in which renewed endocarditis followed another attack of rheumatism.

The Secretaries have asked me to narrate briefly two or three cases. I select three at random. No. 62 in the list, I. B., a strong vigorous schoolboy, æt. 14, who had never before had rheumatism, admitted November 12, 1897, with acute rheumatism and chorea; temperature 102°; pulse 122. Cardiac dulness normal; slight epigastric pulsation; distinct systolic bruit at the apex; accentuation of the second sound at the pulmonary valve; cloud of albumen; profuse acid perspiration. 16th, pain almost gone, temperature normal; cardiac bruit louder. December 6, bruit getting soft. 19th, bruit became variable, and after this date finally disappeared. The boy went home, was kept under observation, all active exertion being interdicted; he remained well until the middle of February, 1898, when he was again attacked by acute rheumatism. On readmission the heart was normal. On March 1 the systolic mitral bruit with second pulmonary accentuations were again heard; uric acid in considerable amount in the urine; some chorea manifest; temperature 101·2°. The patient soon lost the rheumatic pain, and later the chorea, but the cardiac bruit remained long, fully two months; at length it yielded, and his heart became apparently normal. I examined him three weeks ago. I thought there was slight hypertrophy,

but no bruit, no accentuation of second pulmonary sound, not the least dyspnœa; he had grown half a foot, was working hard at a laborious trade. The next case, No. 74 in my list, was not a case of acute rheumatism at all; the patient, I. M. W., æt. 21, was admitted to hospital in May, 1899, for Raynaud's disease, when an attack of acute endocarditis occurred. Patient was a pharmaceutical chemist; a distinct systolic bruit at the mitral occurred, with accentuation of the second pulmonary sound; temperature rose slightly at the time, cardiac dulness remained normal.

Under the usual treatment the bruit and accentuation disappeared in about five weeks. The patient was kept quiet for some months. His heart has remained perfectly normal. I had an opportunity of examining it a fortnight ago.

Case 83. M. C., æt. 25, wardmaid in the Infirmary. Has had no rheumatism previously. Attacked by pain in the knee and other joints on November 27, 1899, and admitted into my ward on December 4. A severe attack of peliosis rheumatica had occurred. Temperature 102·3°. The condition of the skin and the distress occasioned by it, occupied our attention so completely that we did not examine the heart so regularly as usual; on the 12th we discovered that a loud and distinct bruit was audible at the apex, and was conducted into the axilla. There was no increase of dulness; the second pulmonary sound was accentuated. The condition of the skin—sufficiently bad to begin with—was made worse below the clavicle by the blisters for a time. Under treatment fever and pain disappeared, later the peliosis gradually subsided also, but the bruit remained loud and distinct until about January 10, when it began to soften and to intermit. About the 18th it was generally audible when the patient was recumbent, but disappeared when she sat up. On the 25th it had disappeared altogether. On the 27th we failed to hear the previously marked accentuation of the second pulmonary. She went home on February 8 with an apparently sound heart, although a peculiar tremor suggestive of a thrill was perceptible if she exerted herself; this of course she was forbidden to do. I have examined her heart repeatedly since, and it appears absolutely sound, the tremor being quite absent.

In conclusion, there are two principles arising out of this investigation which I should like to propose for your consideration.

The first is this: I think we should regard each case of

acute rheumatism, not merely as *rheumatism*, but first and before everything else, as *a case of impending peril to the heart*, endangering the whole future life of the patient.

The mere rheumatism *per se*, the pain, fever, the general discomfort, with the means at our disposal we can usually soon mitigate, and the patient will after a few weeks' care recover; all this is quite secondary in importance to the great question, will the heart escape damage? The physician should, when the organ is sound at the beginning, examine it almost daily during the course of the complaint—making its condition his chief concern.

The second is the following:—In acute rheumatic endocarditis, as indeed in the non-rheumatic forms, there is a stage during which the disease is usually curable; in adults it is not a long stage, probably only two or three weeks in duration, and if it be suffered to pass by without the needful care and treatment it frequently, if not generally, leads to organic disease; in children the period during which repair is possible is much longer. In children, and to a less extent in adults, by persistent rest alone some cases recover, and a very small number indeed even without it.

If rest be combined with stimulation of the natural reparative process by the method I have suggested, or some other method (for example, a friend of mine stimulates the thoracic skin by the continued inunction of the red oxide of mercury, and he has results like mine), if also one of the iodides be given, or small doses of mercury, in the course of three, four, five, or six weeks, sometimes earlier sometimes later, the bruit will be heard to soften, it will by degrees become intermittent and variable, there will come a period during which it may be heard when the patient is recumbent but disappears when he sits up, at length it will vanish altogether, and so also will the accompanying accentuation of the second pulmonary sound. He will, of course, need to avoid for a time all vigorous exertion, until the damaged and enfeebled cusps regain their normal power of resistance. In general the earlier the treatment follows development of symptoms, the earlier will be restoration, but the severity of the rheumatic attack is also a determining factor.

I cannot expect you to accept this statement on my word alone, and I do not ask you so to accept it, for we all know how easy self-deception is in matters of therapeutics, but I do venture to ask you to try this method for yourselves.

Where it is tried the only fear is of unsuitable cases being

selected, or, if that mistake is not made, of there being a lack of perseverance. In my most successful cases weeks usually pass without the least improvement, and the tendency is great, particularly if one is new to the method, to give up all hope of amendment, and to yield to the patient's natural desire to resume active exertion, when he feels himself perfectly well.

I cannot but hope and believe that if this method were accepted and universally adopted by the profession, the number of cases of chronic valvular disease in our population would to some extent diminish. Personally I have the satisfaction of knowing that the instances in which valvular mischief has originated in my practice during the last fifteen years is comparatively small, greatly less than before I practised the methods described.

Abstract of Eighty-five Cases of Acute Endocarditis, showing Results following the Treatment described, in illustration of Dr. Caton's paper.

No.	Initials.	Age.	Date of admission.	Date of discharge.	Chief physical signs of cardiac disease.	When first observed.		Apparent condition of heart on discharge.		Remarks.
			1884	1885						
1	I. M.	26	Nov. 17	Jan. 30	Mitral bruit	—	Dec. 9	—	Mitral disease	Severe case: resisting salicylates. Long continued.
			1885							
2	J. M.	12	June 15	July 13	Aortic systolic bruit	On admission	—	Normal	—	—
3	G. M.	18	Jan. 6	Feb. 23	Mitral bruit	—	Jan. 26	,,	—	—
4	H. R.	19	Jan. 22	Feb. 19	,,	—	Jan. 25	,,	—	—
5	S. T.	21	Feb. 23	April 14	,,	—	March 9	,,	—	Heart affected in relapse on 9th March.
6	M. A.	16	March 3	March 29	,,	On ad.	—	,,	—	—
7	M. W.	49	March 13	April 30	,,	,,	—	,,	—	—
8	E. L.	19	March 27	May 10	,,	,,	—	,,	—	—
			1888							
9	J. C.	34	April 23	May 24	Aortic systolic bruit	—	April 30	,,	—	—
10	A. C.	19	May 10	June 14	Mitral bruit	On ad.	—	,,	—	—
			1889							
11	L. P.	17	Jan. 10	Feb. 6	,,	,,	—	—	Mitral d.	Left hospital too soon.
12	A. M.	21	May 9	May 22	,,	,,	—	—	,,	—
13	M. J.	66	June 7	July 12	,,	,,	—	Normal	—	—
			1890							
14	M. McC.	16	June 4	July 16	,,	—	June 20	,,	—	—
15	C. C.	21	June 24	Aug. 18	,,	On ad.	—	,,	—	Pericarditis.
16	C. B.	19	Dec. 17	Mar. 23,	,,	,,	—	,,	—	—
			1891	1891						
17	A. J. D.	29	Feb. 7	March 14	,,	—	Feb. 10	,,	—	Severe case: violent delirium.
18	M. G.	14	May 13	June 11	,,	On ad.	—	,,	—	—

Abstract of Eighty-five Cases of Acute Endocarditis—continued.

No.	Initials.	Age.	Date of admission.	Date of discharge.	Chief physical signs of cardiac disease.	When first observed.		Apparent condition of heart on discharge.		Remarks.
			1891							
19	A. G.	18	June 13	July 13	Mitral bruit	—	June 17	Normal	—	—
20	R. S.	13	Nov. 27	Dec. 31	,,	On ad.	—	—	Mitral d.	Bruit much diminished in loudness.
21	I. H. T.	18	Dec. 20	Dec. 31	,,	,,	—	Normal	—	Went out too soon. Bruit absent.
			1892							
22	T. P.	28	Oct. 16	Nov. 28	,,	,,	—	,,	—	Pericarditis.
23	T. T.	26	Nov. 18	Dec. 7	,,	—	Nov. 23	,,	—	—
			1893							
24	A. F.	28	Jan. 18	Feb. 10	,,	—	Jan. 22	,,	—	—
25	Readmitted		May 10	May 30	,,	On ad.	—	—	Mitral d.	—
26	M. R.	21	Jan. 25	Feb. 19	,,	—	Jan. 26	Normal	—	—
27	E. P.	35	March 11	April 20	,,	—	March 14	,,	—	Pericarditis.
28	E. R.	30	May 17	June 23	,,	—	May 23	,,	—	—
29	D. H.	19	July 19	Sept. 17	,,	—	July 21	—	Mitral d.	Bruit disappeared, recurred in relapse of rheumatism on August 21st, and persisted, notwithstanding treatment.
30	M. McK.	16	June 9	July 7	,,	On ad.	—	—	,,	—
			1894							
31	A. D.	42	Jan. 11	March 3	,,	,,	—	Normal	—	—
32	E. P.	19	May 2	May 30	,,	,,	—	,,	—	—
33	E. J.	17	July 4	Aug. 13	,,	—	July 16	,,	—	—
34	R. E.	15	July 4	July 28	,,	On ad.	—	,,	—	Pericarditis.
35	M. F.	18	June 14	Aug. 9	,,	,,	—	—	Mitral d.	—
36	E. K.	24	June 25	Oct. 3	Mitral bruit and aortic diastolic bruit	,,	—	—	,,	Had three successive attacks of rheumatism in hospital. Aortic bruit disappeared.
				1895						
37	M. A. H.	22	Dec. 8	Jan. 18	Mitral bruit	,,	—	Normal	—	—

38	I. B.	38	March 1	April 10	Mitral bruit	On ad.	—	Normal	—	—
39	J. G.	27	Oct. 17 1895	Dec. 6	„	„	—	—	Mitral d.	—
40	R. P.	12	Feb. 6	March 9	„	—	Feb. 19	Normal	—	Chorea.
41	N. G.	43	Jan. 16	March 18	„	On ad.	—	„	—	—
42	A. P.	18	March 4	May 15	„	—	March 5	„	—	But the rheumatism came on again in September.
43	Readmitted		Oct. 1	Nov. 19	„	On ad.	—	—	Mitral d.	—
44	J. G.	14	July 17	Sept. 16	„	„	—	—	„	—
45	I. C.	23	Oct. 12	Dec. 19 1896	„	—	Nov. 11 (?)	—	„	Had also enteric, pneumonia, and pericarditis.
46	R. C.	15	Nov. 20	Jan. 25	„	—	Nov. 26	Normal	—	Chorea.
47	H. G. A.	29	Nov. 23 1896	Jan. 3	„	On ad.	—	„	—	But rheumatism came on again in February.
48	Readmitted		Feb. 19	April 16	„	„	—	„	—	—
49	H. H.	16	Feb. 5	April 9	„	„	—	—	Mitral d.	Bruit very slight.
50	F. M.	9	June 29 1895	Aug. 18	„	„	—	—	„	Bruit less distinct than on admission.
51	M. D.	14	Dec. 11 1896	Jan. 13	„	„	—	Normal	„	—
52	I. B.	20	March 7	April 23	„	„	—	„	—	—
53	I. T. J.	14	Feb. 28	April 11	„	„	—	„	—	Chorea.
54	P. R.	38	March 18	April 23	„	—	March 23	„	—	—
55	M. L.	17	June 21	July 25	„	On ad.	—	„	—	—
56	A. H.	19	Aug. 5	Nov. 21	Mitral bruit (presystolic and systolic)	—	Aug. 13	„	—	—
57	M. S.	18	Nov. 28 1897	Dec. 30	„	On ad.	—	„	—	—
58	F. McD.	18	Feb. 20	May 20	„	„	—	„	—	—
59	A. T.	19	March 15	April 29	„	„	—	—	Mitral d.	—
60	C. K.	19	Sept. 24	Oct. 18	Mitral bruit (presystolic and systolic) aortic systolic	„	—	Normal	—	Bruits all disappeared.

Abstract of Eighty-five Cases of Acute Endocarditis—continued.

No.	Initials.	Age.	Date of admission.	Date of discharge.	Chief physical signs of cardiac disease.	When first observed.		Apparent condition of heart on discharge.		Remarks.
			1897	1898						
61	I. S.	28	Dec. 14	Feb. 21	Mitral bruit	On ad.	—	Normal	—	—
62	I. B.	14	Nov. 12	Jan. 12	„	„	—	„	—	Chorea. Rheumatism recurred in February.
			1898							
63	Readmitted		Feb. 25	June 2	„	—	March	Became normal later	—	Slight hypertrophy remains in 1900.
64	I. E. D.	14	Feb. 1	July 4	„	On ad.	—	Normal	—	Bruit disappeared, but recurred during a relapse of rheumatism; disappeared again after prolonged treatment and rest.
65	A. T.	34	June 17	July 20	„	„	—	—	Mitral d.	—
66	J. T.	24	July 12	July 26	„	„	—	Normal	—	Left hospital too early.
67	F. G.	26	Sept. 16	Nov. 8	„	—	Sept. 18	„	—	—
			1899							
68	L. E.	35	Jan. 24	April 24	„	On ad.	—	„	—	Pneumonia.
69	A. G.	23	March 24	May 5	„	„	—	„	—	—
70	R. G.	8	April 27	June 11	„	„	—	—	Mitral d.	—
71	I. C.	30	April 4	May 29	„	—	April 6(?)	Normal	—	—
72	Mrs. S.	—	May 23	June 19	„	—	May 27	„	—	—
73	T. C.	19	May 16	July 30	„	On ad.	—	—	Mitral d.	—
74	I. M. W.	21	May 30	July 19	„	—	June	Normal	—	Had Raynaud's disease. No acute rheumatism.
75	I. W. J.	33	June 16	Oct. 19	„ Mitral on admission Aortic came on in hospital			— Mitral and aortic regurgitation		Had repeated attacks of rheumatism in hospital.
76	R. H.	15	June 27	July 25	„	On ad	—	Normal	—	Chorea.

77	T. B.	17	July 11	July 29	Mitral bruit	—	June 17	Normal	—	Left hospital too soon.
78	M. F.	26	Sept. 12	Oct. 23	„	On ad.	—	„	—	—
79	M. E. W.	18	Oct. 24	Dec. 28	„	—	Oct. 22	„	—	—
80	M. R.	25	Dec. 19	Jan. 26	„	—	Jan. 10	„	—	—
81	J. M.	21	July 9	Aug. 17	„	On ad.	—	—	Mitral d.	Pericarditis. Left hospital too soon against advice.
82	M. A. G.	52	May 12 1899	June 19 1900	„	—	May 23	Normal	—	Pleurisy.
83	M. C.	25	Dec. 4	Feb. 8	„	—	Dec. 14	„	—	Peliosis rheumatica.
84	I. G.	18	Dec. 12 1900	Feb.	„	On ad.	—	„	—	—
85	T. O.	14	Jan. 26	—	„	—	Jan. 27	?	?	Still in hospital.

Summary of Results.

(1) Cases in which signs of valvular disease were present on admission: fifty-three in number. Of these thirty-five left hospital with heart apparently sound; eighteen left hospital with valvular disease.

(2) Cases in which valvular disease commenced in hospital: thirty-two in number. Of these twenty-eight left hospital with heart apparently sound; three left hospital with valvular disease; one still under treatment.

XIX.—*A case in which the attempt was made to remove a Dermoid Tumour which, growing in the Spinal Canal, pressed upon the Spinal Cord.* By W. Hale White, M.D., and A. D. Fripp, M.S. *Read March* 23, 1900.

OPERATIONS for the relief of symptoms produced by the compression of the spinal cord by tumours within the spinal canal are so rare, that the following case seems worthy of record.

Mr. W., æt. 26, who had been, before his health gave way, a fine athlete, was sent to Dr. Hale White by Dr. Astley Roberts, of Eastbourne, in April, 1895. He had come to Eastbourne to convalesce from influenza, with which he had been attacked many times. Dr. Roberts found several of his cervical and axillary glands much enlarged, and also discovered dulness and harsh breathing at the apex of the right lung. Shortly after this date the patient left Eastbourne, and put himself under the care of Mr. T. H. Smith, of Alcester, who brought him up to town on several occasions to see Dr. Hale White, and once Sir Samuel Wilks also saw the patient. The pulmonary signs gradually disappeared, and the opinion in which all agreed was that he was suffering from Hodgkin's disease. He was treated with 10 minims of Liquor Arsenicalis thrice a day, and improved considerably in health. Most of the affected glands diminished in size, and although fresh ones became evident, they did not attain the size reached by those which had been affected before he began to take the arsenic.

On March 25, 1899, Dr. Hale White was asked to go and see the patient with Mr. A. F. Wilson, under whose care he then was. The history then obtained was that since 1896 the patient had taken arsenic regularly, and that although some newly enlarged lymphatic glands in various parts of his body had from time to time appeared, yet they had disappeared again, so that it might fairly be said that under the arsenic the Hodgkin's disease had been held in check. His general health remained fairly good until the middle of January,

1899, when he began to complain of loss of power in the legs; this got a little better, but became worse again early in March and was soon absolute. When seen on the 25th March there was no sign of the old trouble at the apex of the right lung, the lymphatic glands of both groins and the left axilla were greatly enlarged, the biggest were as big as a walnut, and formed in one groin a mass the size of the fist. He had complete paralysis of both lower extremities, but no precise history could be obtained as regards the order of invasion of the muscles. There was no muscular wasting. His breathing was almost entirely diaphragmatic, the right hand was considerably stronger than the left. Sensations of pain, touch, and temperature were completely lost up to a nearly horizontal line on the front of the body three quarters of an inch below the nipples. At the back the complete anæsthesia extended about as high as in front, but its upper margin was ill defined. There was no hyperæsthetic zone. He had severe pains shooting down both intercosto-humeral nerves, and round the upper part of the chest along the course of the second intercostal nerve. When the pain was very severe it occasionally reached the fingers. The sensation of the arms was in all respects normal, except that perhaps there was a little hyperæsthesia in the area of distribution of both intercosto-humeral nerves. The lower limbs were in a state of spastic rigidity liable to severe exacerbations in which the flexors overcame the extensors. This rigidity distressed him very much. The knee-jerks and plantar reflexes were exaggerated. There was no ankle-clonus, no patellar clonus, no priapism, nor any cremasteric or abdominal reflex. There was great superficial tenderness over the second, third, and fourth dorsal spines. He had incontinence of urine with considerable cystitis. He also had most obstinate constipation, and much abdominal distension. His pulse was usually about 120, and his temperature between 100° and 101°. The buttocks were a little red. The pupils and optic discs were normal. There was no irregularity nor unnatural prominence of the bones to suggest caries of the spine, nor was there any evidence that an aneurism was eroding it.

From these evidences it seemed clear that there was either a collection of fluid, or a new growth in the spinal canal, which irritated the second dorsal nerve enough to cause pain along it, but not enough to affect the pupil or to produce much hyperæsthesia, which compressed the cord a little below

this, and which caused secondary degeneration of the pyramidal tracts below the point of compression. There was no evidence that this something was in any way connected with the bones, nor was there any evidence that it was syphilitic. It was difficult to see how it could be connected with the Hodgkin's disease, but we did wonder whether some lymphatic overgrowth in the posterior mediastinum could be growing along the roots of the spinal nerves, and so into the spinal canal. A careful physical examination of the chest, however, failed to reveal any evidence of such an overgrowth.

The position of affairs was fully explained to the friends and to the patient; it was pointed out to them that if left alone the case could only have one end, and that probably a very painful one, from bedsores, cystitis, and pyelitis: but, on the other hand, although any operation was serious yet it was possible that the pressure on the cord might be removed even if the tumour could not be taken away, and that at his age it was probably not malignant. After hearing this the patient and his friends decided that a surgeon should see him, and accordingly on March 29th Mr. Fripp joined Mr. Wilson and Dr. Hale White in consultation. The patient's symptoms were unchanged except that the line of the upper limit of anæsthesia was a little nearer the nipple. It was agreed that an exploratory laminectomy should be performed, and it was done on the afternoon of the same day. As there was absolutely no evidence of syphilis the case was clearly too urgent to admit of delay for an experimental course of iodide of potassium.

Mr. Rowell gave the anæsthetic, and a more difficult case for administration we have seldom seen. The intercostal muscles were paralysed, and the action of the diaphragm was much interfered with by the enormous distension of the abdomen, and by the semi-prone position required for operation. Mr. Eason assisted.

Through a median incision seven inches long the spines and laminæ were, by means of the knife, quickly denuded of the muscles attached to them. The periosteum was scraped aside as completely as was compatible with the need for rapidity. It was not found necessary to make any transverse incision through the aponeurosis. Hæmorrhage was brisk, but soon yielded to pressure, forceps, and packing. No ligatures were applied.

The patient was a large-boned man, and the removal of

the second, third, and fourth dorsal arches was hard work. The laminæ were divided by bone forceps of various shapes, of which Horsley's bent pattern was the most useful. Instead of the layer of fat and vessels which is usually exposed as soon as the spinal canal is opened, we saw a grey mass of growth bulging out of the whole length of the deficiency in the spinal canal, and traceable round the two sides of the cord as far as retractors could enable us to see. This mass of growth was incised in the middle line to a depth of about a quarter of an inch, but nothing was seen of the dura mater. It hardly bled at all. Knowing that lymphadenomatous growths were present in other parts of the body we thought this mass must be of the same nature. The condition of the patient precluded such a prolongation of the operation as would obviously be necessary were any attempt made to get to the limit of this apparently unlimited mass of diffused growth. So pieces of it were snipped off with scissors, and the patient put back to bed as quickly as possible, the administration of the anæsthetic having lasted just an hour. Many sutures of salmon gut were used to bring the surfaces of the wound together. A small drainage tube was laid along the entire length of the bottom of the wound, and brought out at the lower end.

Aseptic pads were used for dressing, and the deeper were so arranged that they were not disturbed when the drainage tube was removed forty-eight hours later.

The patient quickly regained consciousness, and made a good recovery from the operation.

All the stitches were removed on the eighth day, primary union having taken place.

Several sections of the pieces of tumour removed were examined microscopically, and it was found to consist chiefly of fibrous tissue with a quantity of recent inflammatory cells. But the surface of the tumour showed a series of perfect layers of epithelium exactly like skin, together with sweat glands and hair follicles. We showed the sections to Mr. Targett. He quite agreed with us in considering the tumour to be a dermoid.

We told the patient and his friends that the tumour was unconnected with Hodgkin's disease, and that it was not malignant.

On May 16, 1899, the patient's friends asked Dr. Hale White to see him again with Mr. Wilson to advise if anything could be done with a view to further relieving him.

His condition at this date—that is between seven and eight weeks after the operation—was as follows:

There was a healthy scar. The pulse was usually between 80 and 100, and the temperature was normal. He could occasionally move the toes and legs slightly. There was no clonus. The exaggeration of the knee-jerks was less, and the distressing rigidity had disappeared. There was almost complete return of all forms of sensation down as far as the groin, and there was slight, but very slight, appreciation of all forms of sensation in the lower extremities. The pain down the intercosto-humeral nerves had gone. The patient had control of his bladder, the cystitis had disappeared. The abdominal distension and constipation were much less. There was no pain in the back, and the chest moved better on respiration. Unfortunately in spite of the great care on the part of the nurse there was a large bedsore over the sacrum and another over the right buttock. His condition was so far satisfactory in that it showed that the taking away of the laminæ had removed some of the symptoms of pressure on the cord.

Considerable as had been the relief it appeared to us that as (1) the return of power and sensation in the legs was trifling, (2) we knew that the tumour was left behind, (3) the bedsores were extensive and the sacrum was exposed, the patient's only chance of recovery lay in a further attempt being made to remove the growth, the nature of which was now revealed. At our request Mr. Fripp again saw the case, and as he concurred in our view, and as the patient and his friends wished the attempt made, after the desperate position had been fully explained to them, a second operation was performed on May 20, 1899.

Mr. Rowell again gave the anæsthetic, and Mr. Eason assisted.

The conditions were just the same as at the first operation, except that the breathing was somewhat less embarrassed. As soon as the skin was divided by the median incision the growth presented, the erectores spinæ having evidently been pushed to either side by it as it bulged backwards out of the deficiency made in the spinal canal at the first operation. It was not encouraging to our hopes of getting to the limits of the growth to thus find that it was of such a nature that it could effect so wide an extension in so short a time.

Having burrowed down the side of the growth till the rough roots of the second, third, and fourth laminæ could be

felt, we first worked upwards and removed the first dorsal arch, then downwards and removed the fifth and sixth. Still there was no indication of any limit to the growth, so we cut some of it away, and closed the wound as before. The hæmorrhage was less than at the first operation.

Again the duration of the anæsthesia was about an hour, and again the patient recovered consciousness a very few minutes after the conclusion of the operation, and answered cheerfully questions put to him. He appeared to have borne the operation well, but eight and a half hours afterwards he suddenly died.

No post-mortem examination could be obtained. Our regret that the tumour when reached was not removable is tempered with the assurance we feel that the attempt to remove it was justifiable.

XX.—*A Report of Ninety-nine Cases of Angular Deformity from Pott's Disease treated by Manual Rectification, including the Results of Twenty-five Cases brought before the Society in November*, 1897.

By ROBERT JONES and A. H. TUBBY, M.S. *Read March* 23, 1900.

IN November, 1897, we were privileged to bring before this Society an account of twenty-five cases of angular deformity of the spine upon which we had operated during the preceding five months. The objects of that preliminary communication were to emphasise the following points, that—

1. Rectification of the deformity is not *per se* an immediate danger to life.

2. Paraplegia does not necessarily occur by reason of the operation.

3. Many curvatures of the spine by the exercise of some pressure can be lessened and obliterated.

But we were not then in a position to make any definite statements as to the more remote complications which resulted from the procedure, such as the dissemination of tubercle, the incidence of abscess, and the onset of late paraplegia. Nor could we say if the gap in the line of the vertebral bodies were replaced by material sufficiently firm to maintain the body in the erect position without instrumental support after a lapse of some years.

At the time of reading our former paper we, at the request of some members of this Society, promised to publish the results of these cases, and to show as many of them as possible.

We are now able to give statistics of our results in these twenty-five cases, to detail further experiences, and to exhibit fourteen patients who are now walking.

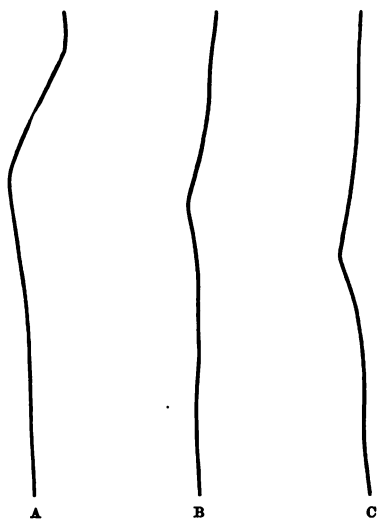
In justification of our early attempts upon the spine we adduced the following reasons for not yielding to what may be termed the accepted pathological forecasts:

Firstly. In the immediate rectification of quiescent tubercular joints elsewhere, we had experienced an unexpected amount of success, quite at variance with the usual teaching in this country, but lately accepted in America.

Secondly. In the application of the plaster of Paris jacket by Sayre's method with suspension, a certain amount of spinal extension occurred, and many surgeons acknowledge that the deformity was temporarily diminished without any evil results occurring.

Thirdly. We were conscious that the mere presence of spinal deformity, with its immediate evil consequences upon the shape of the chest, and more remote consequences upon

FIG. 4.



Outlines of spine of child æt. five years. Outline A represents the curve at the beginning of treatment; outline B the curve about one year after the first straightening; outline C the condition of the curve in March, 1900, two and a quarter years after commencement of treatment.

the physiology of the lungs, heart, and great vessels, tended to shorten life.

It has been shown by Neidert* that of all cured cases of Pott's disease attaining adult life, the expectation of life was not more than forty-nine and a half years. We felt that if this method of rapid reduction were free from danger and permanent in its effects, we were justified on this ground alone in making a trial of it.

Fourthly. We were assured that the more the curve

* See *Inaugural Dissertation*, Munich, 1886.

was reduced the greater was the ease of fixation, and the difficulties in limiting its increase became lessened; for the straighter the spine the less is the leverage arising from the weight of the shoulders and head upon the weak point in the spinal column, and the more effectively can the spine be controlled.

Fifthly. We thought that by this method pressure on the cord might be removed, and paraplegia be cured.

We have also endeavoured to ascertain if the frequency of acute tuberculosis and abscess is greater in cases in which the spinal reduction is carried out.

The difficulties of the investigation have been great. Not only is there considerable physical labour involved, and much time consumed in readjusting the apparatus, and seeing the children at sufficiently frequent intervals, but we have had to contend with the general neglect of poor parents who often allow their children to remain in such an insanitary state that the use of the splint has to be discontinued for a time in order to get the child wholesome.

As a result also of the neglect of parents to bring the patient to the hospital at the time stated, pressure sores have formed, and delay has been occasioned while they healed. Then the splints have been removed, either temporarily or permanently, by parents on their own responsibility for various reasons; and lastly we have met with disappointment in the permanent desertion of the patient, the parents often being irritated with the tedium of the after-treatment or unable to spare the time requisite for bringing the child at the proper intervals of not less than a month for inspection, readjustment, and reapplication of the splint. Or the patient has sought advice at other hands, and an inefficient poroplastic jacket has been recommended for high or mid-dorsal curves.

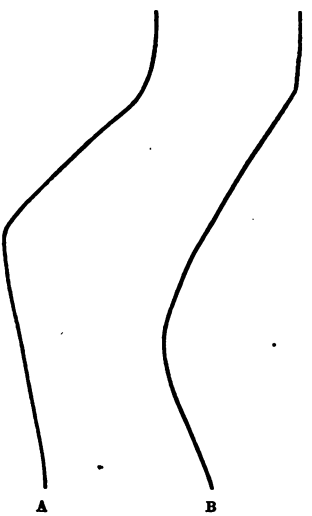
We therefore feel that the responsibility of the surgeon in undertaking to treat hospital cases of spinal disease on these lines is very considerable, and he should weigh well the account of our difficulties before beginning a case, and should make up his mind to see it through. We know that the pressure of other and often more interesting work is likely to seriously interfere with the desire to fully superintend a number of these cases, and the further difficulty of always enlisting the services of enthusiastic and trained assistants is somewhat considerable, although we have to acknowledge that we have been particularly fortunate in our assistants, and make grateful acknowledgment of their services. We

have now to present to you the results of two series of cases: a first series of twenty-five, spoken of in our paper in November, 1897, and a second series of seventy-four cases operated upon since that time, from two and a half years to within a few weeks of the present time. Taking, then, the first series of twenty-five cases we have the following results:

5 have died.
4 have discontinued treatment.
15 are alive and well and walking.
1 is recumbent, and is thin, sickly, and prone to develop pressure sores.

25

FIG. 5.



Two tracings of the spinal curvature from a case treated by this method. A represents the condition at commencement, and B that at the end of two years after attempts to rectify the curve.

To go more fully into these statistics. As to the causes of death:

1 case died with persistent vomiting four days after operation; but at the post-mortem examination, a somewhat imperfect one, no lesion in the abdomen was discoverable beyond some enlarged mesenteric glands.

1 died of tabes mesenterica fifteen months after operation.
1 died of pulmonary tubercle sixteen months after the last stretching.
1 died of diphtheria two years after; and
1 died of intestinal obstruction twelve months afterwards.

Thus of the twenty-five cases, one death may fairly be said to be directly associated with the operation, and two deaths have occurred from complications which are known to occur from Pott's disease. Taking the cases of Billroth and Menzel, Jaffé and Mohr, we find that they estimate the death rate from Pott's disease to be 25 per cent.; whereas our death rate in two and a half years was only 12 per cent. in these twenty-five cases from the results of Pott's disease. That it has been so small we believe to be due to the careful supervision of our cases, and the efficient fixation they have been submitted to.

Four cases discontinued treatment. One child who was doing admirably went to Holland, another to Tyrone, one took scarlet fever, and the splints were removed with resulting recurrence; and another removed the splint because of bronchitis.

Four of the cases developed abscess, and of these two have healed, and one case became paraplegic eight months after the operation but has since recovered.

Five of the twenty-five cases recovered from paraplegia as the immediate result of the operation, and in only one case did it recur, and this in a child who subsequently died from intussusception. In respect of paraplegia the operation may be regarded as a successful one, and containing greater possibilities in inveterate cases of paralysis than laminectomy.

In five of the twenty-five cases the curvature was practically obliterated, and remains so with a spinal support, and the curve was much improved in ten.

As was before mentioned, fifteen of the cases now under our observation are walking, and one is recumbent.

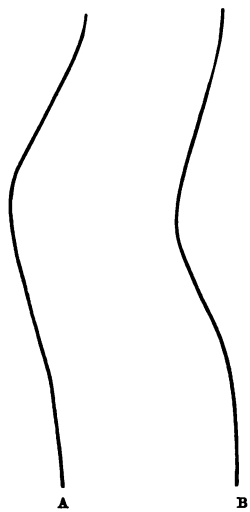
For the purposes of comparison and appreciation we now speak of twenty-five cases of spinal deformity who refused manual rectification, and came under our observation about the time our first series were operated upon. They were in fact control cases.

Of these three have died, all from tubercle; four have developed abscess, and the abscesses have healed in two, and remain still open in two cases; four have become paraplegic,

of whom two have recovered; and in seven cases the deformity is very marked, and is not improved in four. Three discontinued treatment.

The number of reductions in the twenty-five cases operated on was seventy-three in all; the greatest number in an individual case was seven, and the least one.

FIG. 6.



Two tracings of curves from a child whose spine was rectified by manual pressure and extension. The improvement in the curve B over A is well marked.

	With operation.	Without operation.
Death from all causes	5 (3 due to complication of Pott's disease)	3
Tubercular disease	3	3
Abscess	4	4
Paraplegia	1	4
Deformity decreased	15 (17 including the patients who went away.)	Markedly increased in 7, not improved in 4.

It therefore seems to us that so far in these twenty-five

cases operated upon, the number of deaths and the incidence of abscess, the results in these cases are by no means discouraging. Whereas the recoveries from paraplegia, and the results as to obliteration or reduction of the curvature which we are able to show you to-night, are evidence that with properly selected cases and in judicious hands the method has a future before it.

We now speak of our second series of seventy-four cases, operated upon since the above date until within a few weeks of the present time.

7 of these have died.
29 cases are walking.
28 cases are recumbent.
10 have discontinued treatment.

74

Of the 7 deaths:

1 died from empyema four weeks after the operation of reduction.
1 „ „ general tuberculosis thirteen days after operation.
1 „ „ „ „ two years after operation.
1 „ „ pulmonary phthisis eighteen months after operation.
1 „ „ pneumonia two years after operation.
1 „ exhausted by discharge from an abscess.
1 „ six months after a reduction for paraplegia without relief, in whom laminectomy was performed, but he succumbed to diarrhœa one month after laminectomy, although considerable power had returned.

10 either removed the splint without permission or discontinued treatment.

4 removed the splint with our permission: 1 because of constant vomiting and pain while extension was being kept up, but who recovered when the extension was removed and he was placed in a simple support; 1 on account of extensive bedsores, the result of neglect, whom we subsequently kept recumbent; 1 at the desire of the parents eight months after operation, because they could only bring the child at long in-

tervals, and the curvature when the splint was removed was found to be considerably diminished. 1 because of epilepsy; the child is now doing well in a posterior support.

3 were operated on with an abscess present: 1 because of paraplegia, which was improved but not entirely removed by the operation; in 1 a mid-dorsal curve of a very angular variety was operated upon, and the abscess was noticed the day after operation; and in 1 severe girdle pains were present, having lasted three months, which disappeared in a week. The curve is diminished. Although the abscess is discharging the child looks healthy.

13 cases have become complicated by abscess after operation at periods of three to eighteen months, and of these 9 have healed.

7 cases have recovered from paraplegia, or it has been relieved and remains well.

In 38 cases the curvature has been lessened, and remains so. In 16 cases the curvature has either not improved or has become somewhat worse, not necessarily because of the operation, but certainly in 8 of them on account of an unauthorised removal of the splint. In no case where treatment has not been abandoned is the curve increased.

As before stated, 29 are walking and 28 are recumbent.

As to the present condition of these recumbent cases four are ill from tubercle, three have discharging abscess, and in three patients the abscess has closed.

To sum up the whole of our ninety-nine cases to this date:

*12 have died in three years, or a little less than 12 per cent. (With this should be compared the death rate, which is said to be 25 per cent. in cases which have not been operated upon.†)

15 have discontinued treatment.

44 are walking, some with and some without splints.

28 are recumbent.

—

99

* Of these twelve, two were due to causes not arising from the spinal lesion, as, *e.g.*, diphtheria and intestinal obstruction.

† But it is fair to state that the authors quoted on p. 150 do not name the period over which their observations extended. Presumably it was longer than two and a half years.

17 have developed abscess, and in 11 of these the abscess has closed. This compares favourably with 20 per cent. in unoperated cases.
12 have recovered from paraplegia.
6 are ill from tubercle.
In 4 the curvature has been obliterated or improved.
In 16 the curvature is not improved.
15 patients have discarded the support, or discontinued treatment.

We would now advert to the method we have followed, and primarily we would make this observation:

At the time we read our first paper, and probably the idea is now widely existent, that a large amount of force is called for. Such is not the case. We have found in many cases when extension is properly and simultaneously applied, that the mere extension reduces a part of the prominence, and pressure which can be exerted with one hand is sufficient for the purpose at each sitting. Indeed, we have limited ourselves in this way, feeling that if the prominence did not yield to such pressure, any additional force was undesirable, and the case was not suitable for this treatment. We have been much impressed with the fragility of some of the cases, and we have wondered that with so weak a spot in the spine patients have been able to walk about without support. Indeed, the term "forcible reduction" is in many cases erroneous, and we would earnestly deprecate the idea which is put into practice by some Continental surgeons that a large amount of force should be employed. Our plan has been to proceed by stages, being content with the gain obtained at one sitting, and then waiting for a month.

Our methods have not differed in any great degree from those we described in our former paper, although we tried in some cases a form of splint which, while supporting the back, shoulders, and head, allows the patient to walk about. Still we are convinced that a splint which keeps the patient extended in the recumbent position is the proper thing.

As to the length of after-treatment, this should not be less than three years, and possibly longer in some cases.

We may then come to the following conclusions; that—

1. In the danger to life the risk is not great, two cases of ninety-nine having died within a week of the operation, and the death-rate is not so great as in other statistics before the operation was practised. The explanation is that our cases have been kept under observation.

2. That so far from inducing paraplegia, the operation is actually a curative measure, no less than twelve of ninety-nine having been cured of paraplegia.

3. That the probability of abscess formation is not greater than when the spine is left untouched, but rather less.

4. That the dissemination of tubercle is quite as likely to occur when the spine is not operated upon as when it is straightened; therefore that is a negligible risk.

5. There is no risk of flail-like spine. (The cases which are shown this evening are sufficient evidence of that.)

As to when the operation should not be performed, we are agreed that—

1. It should not be performed at all in cervical curves, and not in high dorsal curves unless there be paralysis, because in either kind of curve the leverage of the head is such as to make the avoidance of recurrence a matter of the utmost difficulty and almost impossible.

2. Not in ankylosed spines.

3. Not in very large and angular deformities.

4. Not when there are evidences of tubercle elsewhere, *e. g.* in the glands or skin.

5. Not in children under two years of age, for troublesome symptoms such as vomiting, rise of temperature, are apt to follow, and young children are difficult to keep clean afterwards, and cannot remain quiet in the splint.

6. Not in adults over twenty-two, because of the bony changes which have occurred.

7. Not if abscess be present, unless paraplegia is also co-existent, a comparatively rare occurrence.

We think that it is an operation which should be performed—

1. Only in comparatively strong children, free from abscess and other form of tubercle.

2. That treatment should only be undertaken when the spine is yielding.

3. That small rapidly-forming curvatures in the mid-dorsal and lumbar regions are the best and have the best prognosis.

4. That if large rapidly-forming curvatures in these regions are treated, they should be done in stages with a month's interval between.

5. That paraplegia, other things being equal, is a strong indication.

In many cases the object of treatment is not to obliterate

absolutely all the prominence, as this is often found to be impossible, but to place the spine in as erect a position as possible above and below that portion of curve which experience at the operation shows to be incapable of reduction without severe force. It will often happen that the extremities of the deformity may be reduced, while its centre cannot be forced into the straight line.

A somewhat extended experience has taught the writers that:

1. The cases for the operation must be selected with great care and discrimination, and that it is not an operation to be practised except by those who fully understand the mechanics and pathology of spinal deformities.

2. That personal care of the patients must be unremitting.

3. That above everything else the patients should be kept in as good air as possible after operation, and during the whole after-treatment. The air of large cities is inimical to successful ultimate results in this and in other operative procedures where tubercle is the fundamental cause.

DESCRIPTION OF PLATES II—V.

To illustrate Mr. Jones's and Mr. Tubby's paper on the Manual Rectification of Angular Deformity in Pott's Disease.

Susan M—, æt. 9 years. Operated upon in 1897. The worst curve we have treated. Most of the dorsal vertebræ were involved in a very acute curve. We would *not* undertake such a case in the future, although it has been largely successful, as reference to the photograph will demonstrate.

Operation repeated seven times. Patient previously very feeble of limb, emaciated, and walked with head forward. The curve had existed since the child was two years old, and was steadily getting worse.

No complications after operation, but during last twelve months very ill from congestion of lungs. Can walk long distances, spine firm. Walking six months.

UofM

Susan M. At commencement of treatment.

Adlard & Son, Imp.

Susan M. At commencement of treatment.

Adlard & Son, Imp.

M 40 U

UofM

Susan M. Two years after rectification of spine.

Adlard & Son, Imp.

Susan M. Two years after rectification and continued support.

Adlard & Son, Imp.

Main

DESCRIPTION OF PLATE VI.

To illustrate Mr. Jones's and Mr. Tubby's paper on the Manual Rectification of Angular Deformity in Pott's Disease.

James I—. One of the most pronounced curves we have seen, involving the column from the third dorsal to about the twelfth. It had existed over three years. Previous history was one of invalidism, culminating in what was described by Dr. Stott as "walking like a crab." He was unable to keep on his feet. Left limb contracted and rigid. Right paralysis. Child very thin and cadaverous.

Six times operated upon. On the first occasion we were all alarmed by the loud crunching sound to which we alluded in our first paper.

Curve immensely improved. Walking for some months.

James I. At commencement of treatment.

James I. After two years' treatment by rectification and supports.

Adlard & Son, Imp.

Mau

DESCRIPTION OF PLATES VII—IX.

To illustrate Mr. Jones's and Mr. Tubby's paper on the Manual Rectification of Angular Deformity in Pott's Disease.

Olive T—, æt. 8. Healthy till three years ago. Since then has always complained of her back until twelve months later, when she was laid up. For eighteen months had very severe girdle pains, paraplegia, and was unable to sit in bed. Lumbar abscess present, but not opened.

Operated upon curve, 1897. Operation repeated twice without anæsthetic. For two months was uncomfortable, but afterwards quite easy. Double psoas abscess, both healed.

Curve small and very angular, occupying lower dorsal and upper lumbar vertebræ, now obliterated. Can walk any distance.

UofM

Olive T. Before treatment.

Allard & Son, Imp.

Olive T. After two and a half years of treatment by rectification and supports.

Adlard & Son, Imp.

Mail

Olive T. After two and a half years of treatment by rectification and supports (standing position).

Ballard & Son, Imp.

Mqu

XXI.—*A case of Acute Encephalitis in a Child following Influenza, with Autopsy.* By MARMADUKE PRICKETT, M.D., and F. E. BATTEN, M.D. *Read May* 4, 1900.

THE history of the case is as follows:

M. R., æt. 9, the eldest of four children, was a fairly strong boy, a son of Greek parents, spare, and possessing the usual nervous temperament of that nation.

In January, 1899, the family were all suffering from influenzal catarrh, but of so slight a character that no medical man was consulted, and the boy was allowed to go to his day school as usual, though troubled more or less with a cough. On the 19th he went to a children's party and danced a good deal and enjoyed himself thoroughly, though his cough was troublesome and attracted attention, and it became so much worse during the night that he was kept in bed, and his temperature was found to be 100° F., and some wheezing was observed. On Saturday, January 21, he was better and was allowed to get up, but he seemed weak and tired and his appetite was poor; still, in the evening he brightened up and played with the other children and appeared to be in good spirits. The apparent improvement continued on Sunday, but on Monday morning he did not seem so well, and asked to be kept at home instead of going to school; yet on this day also he played with the rest and appeared as bright and as merry as possible, and expressed himself as eager to go to school the next day. The cough, too, was less, but the temperature does not seem to have been taken. He went to bed as usual at 8 P.M., and after one or two fits of coughing went to sleep, and nothing more was heard of him till Tuesday morning when the French maid went to wake him for his bath. He then spoke to her in French, and said his arm felt funny and that he didn't want his bath. The maid thereupon fetched the nurse, who, finding his arm helpless, sent at once for Dr. Prickett. When he arrived about 9.30 he found the boy hemiplegic. The eyes and head were turned to the left, and the right arm and leg were completely paralysed. He was aphasic but conscious, and understood what was said to him and tried to answer, and cried a little when he found he

could not speak. His temperature was only just above normal, and the pulse about 90 and somewhat excited. All the organs appeared to be healthy. He soon became more drowsy, cried out occasionally, and put his left arm to his head as if in pain, but he could be roused easily, and answered questions by nodding and shaking his head. He was merely kept quiet, fed on milk and beef-tea, which he swallowed easily, while a calomel purge was administered and small doses of iodide of potash. The next morning he was seen by Mr. Victor Horsley, who suggested small doses of salicin, believing the condition was due to the toxins of influenza. He was also seen by Dr. Buzzard the same afternoon. In spite, however, of all treatment he grew rapidly worse, the coma deepened, and he began to take his food with difficulty and to pass his urine in bed. On Thursday the symptoms increased in severity and the coma became profound, and he died on the morning of Friday, three days after the onset of the paralysis.

Post-mortem performed nine hours after death.

The body was just warm, *post-mortem* rigidity well marked. The dura mater was removed together with the skull-cap.

Brain.—The surface of the brain was pale with marked flattening of the convolutions, especially on the left side, and in the parietal region the bulging appeared to be more marked than in the other portion of the brain.

All the sinuses appeared to be perfectly normal, the vessels at the base of the brain were slit up, but no thrombus or embolus could be found in any of the larger vessels. The middle ears were perfectly normal, and the cerebellum, pons, and medulla appeared normal.

The brain was hardened in formalin for one week, after which it was cut by horizontal sections. On examination of these it was seen that at various places in the subcortical region the vessels were very much injected, and stood out in striking contrast to the surrounding tissue. This condition, although most marked on the left, was not limited to this side, but affected the right side also, and especially the centrum ovale just to the right of the longitudinal fissure. The internal capsule and the basal ganglia appeared normal, and there was no softening of any portion of the brain; the lateral ventricles were of normal size, and the fluid in them was perfectly normal. No cultures were made from the brain.

Microscopical examination: method of examination.—After the brain had been hardened in formalin, pieces were re-

DESCRIPTION OF PLATE X.

To illustrate Dr. Prickett's and Dr. Batten's case of **Acute** Encephalitis.

Fig. 1.

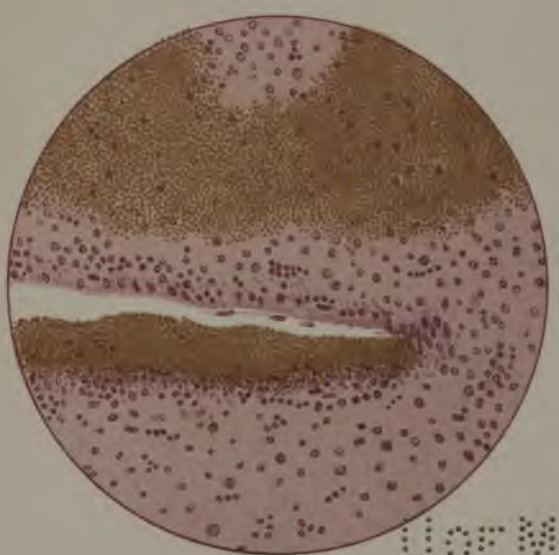


Fig. 2.

Bale and Danielsson, Ltd., Del. and Lith.

moved from the cortex in the frontal, parietal, and occipital regions of both sides, and also from the pons and medulla as well as from the centrum ovale in the situation where the disease appeared most marked; and sections cut from these were stained by Nissl's and by van Giesson's methods. The middle cerebral vessels, both the larger and finer branches, were cut in longitudinal and transverse sections.

Cortex of left side.—Scattered throughout the white matter of the cortex in the frontal, parietal, and occipital regions were numerous small extravasations of blood, mostly in the neighbourhood of vessels, but some independent of them (Fig. 1). The vessels were engorged and filled with blood cells, but there was no organisation of the clot. The vessels in these situations appeared thrombosed. The region of the white matter that was most affected was that which lay immediately below the grey matter of the cortex, and the contrast between the grey matter and the white matter in respect to the punctate hæmorrhage was striking, for although the capillaries in the grey matter were dilated and surrounded by a large number of small round cells, yet there were very few hæmorrhages as compared to the white matter.

Around the vessels and in the neighbourhood of the hæmorrhages there have been exuded numerous small round cells; some of these are of the normal size of leucocytes, but there are others which appear to be much smaller and measure less than ·002 mm.; these small cells would seem to be either blood-platelets or cells undergoing disintegration (Fig. 2).

The examination of the cells of the cortex by Nissl's method showed that the cells were swollen and globular, the nucleus and the nucleolus were for the most part well stained and centrally placed, but the chromophilic substance of the cell was feebly stained and had a fine granular appearance.

This condition was as marked in the occipital and frontal regions as in the parietal, and even in the situations where the congestion was most marked the cells did not appear to be more affected than in the areas where the congestion was less apparent.

Cortex of right side.—The cortex of the right side did not present the extreme congestion that was seen on the opposite side, the vessels and capillaries were full, but there were comparatively few hæmorrhages and but little exudation in the white matter immediately below the cortex. In the centrum ovale, however, on this side the same changes were present as have been described on the left side, viz. small

hæmorrhages, exudation of leucocytes in the neighbourhood of vessels, and thrombosis of the smaller vessels.

By Nissl's method the cells of the cortex in the occipital, parietal, and frontal regions of the right side exhibited much the same appearance as on the left side; the outline of the cells was distinct, with a well-stained nucleus and nucleolus; they were somewhat swollen and globular, and their chromophilic granules had a fine granular appearance.

Medulla.—Sections made through the region of the olive and also below this region showed that there was an increased vascularity, but no hæmorrhages. The cells even in this region did not stain well by Nissl's method.

Cerebral vessels.—Although there was no thrombus in the first portion of the mid-cerebral vessels, yet on tracing them up it was found that both were thrombosed in their finer branches. On microscopical examination the walls of the vessels appeared perfectly healthy, although there was an appearance in some that resembled thickening of the intima; this was probably due to the section being somewhat obliquely cut, or at the bifurcation of the vessel, for on recutting the vessel and taking special precautions to get the section transverse no abnormal feature could be seen.

Summary.—The pathological changes found in this case were as follows:

(1) Engorgement and thrombosis of the vessels of the cerebral cortex with minute extravasations of blood and exudation of leucocytes in the neighbourhood of vessels, situated for the most part in the white matter of the brain immediately subjacent to the grey matter, the condition being more marked on the left side than the right.

(2) A swollen condition of the cells of the cortex as shown by Nissl's method without alteration on the nucleus or nucleolus, the staining of the chromophilic granules being defective.

(3) Thrombosis of the finer branches of the cerebral vessels.

(4) The presence of minute cells arranged in groups of two or more in the neighbourhood of the thrombosed vessels and of the hæmorrhages.

Remarks.—The case above described undoubtedly belongs to those known as acute encephalitis occurring after some acute infective disease, and especially after influenza, and there would seem to be little reason to doubt from the slow and progressive nature of the symptoms that the condition was

primarily due to thrombosis of the smaller vessels; whether such thrombosis was due to changes occurring in the walls of the blood-vessels or to changes occurring in the blood is a question not easy to decide.

The description of the disease, as given by Strümpell in his first paper, refers rather to cases of a less fulminating nature, while in a paper published in 1891 he describes two cases both occurring in adults with high temperature, loss of consciousness, hemiplegia, and death in a few days; and the microscopical appearances correspond with those found in the present case, viz. numerous punctate hæmorrhages into the centrum ovale, with dilatation of the vessels and exudation of small round cells in the neighbourhood of these and of the hæmorrhages.

The close association of the disease with influenza has of late been shown by Lichtenstein, Furbringer, Koenigsdorf, Flatau, and others. Many of these cases, however, had a favourable issue, and in some recovery was complete.

Whether the condition is directly due to the presence of the influenza bacillus in the intra-cranial cavity, or is due to the influence of the toxins formed, is a question that has not been decided. The influenza bacillus has been found in the brain in one case by Pfuhl, but as his case was one of mixed infection, it can hardly be taken as evidence on the point. Oppenheim, in his article in Nothnagel's *System* on "Die Encephalities," states that Nauwerck has obtained the Pfeiffer bacillus from a case of encephalitis.

The observation made in the number of extremely small cells in the region of a thrombosed vessel would suggest that these cells are of the nature of blood-platelets, and it has been pointed out that in such infective diseases as influenza, pneumonia, etc., there is an increase in the blood-platelets, and also it is in these cases that thrombosis is liable to occur (see art. "Thrombosis," *Allbutt's System of Medicine*, vol. vi, p. 177). Oppenheim, in summarising the pathology of the condition, says, "In nearly every case no change could be found in the arteries, and there was no thrombus or embolus; there were numerous small and large hæmorrhages, especially in the neighbourhood of the vessels, and besides these small groups of blood-corpuscles which in cases of longer duration were undergoing disintegration, round-cell infiltration was always present."

It will be noted that in regard to the condition of the vessels, we are not in perfect agreement with Oppenheim; on

the other hand, in the present case, although we believe and have stated that the vessels were thrombosed, it would be difficult to prove that they are not simply engorged, as sufficient time had not elapsed for changes characteristic of thrombosis to occur.

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7. Pfuhl u. Walter. *Ibid.*, p. 82, 1896.
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XXII.—*Three Cases of Removal of Gall-stones from the Biliary Passages* By William Henry Battle. *Read May* 11, 1900.

NOT long ago the removal of stones from the gall-bladder was a procedure of uncommon occurrence. Now that operation is frequently undertaken, and with about as much safety as the radical cure for hernia, when there is no cholæmia.

The removal of stones from the bile ducts is, however, much more rarely required, is an operation involving greater risks to the patient, and usually causes much greater trouble to the operator.

The actual removal of the stones in these cases was done under favourable circumstances, none of the patients suffered from jaundice, there were no adhesions about the ducts obscuring the position of the stones and making their localisation difficult, and the amount of bleeding the result of incision of the duct, was in no case troublesome.

In each case after the position of the stone or stones had been made out gauze packing was employed to prevent any extravasation of bile into the peritoneum around; whilst the employment of a single row of Lembert's sutures of fine silk was found sufficient to securely close the incisions after the removal of the stones.

The cases were admitted under the care of my medical colleagues at St. Thomas's Hospital.

Case 1.—A girl, æt. 14, was admitted under the care of Mr. Hawkins in 1898.

She was an extremely fat but healthy looking girl, who gave a history of intermitting attacks of pain in the abdomen for a period of about six months. This pain had occasionally been of a very severe character, and was referred to a point usually below the umbilicus. The urine had been thick at times and of a dark colour. She had never had jaundice. Examination of the left lumbar region caused a complaint of tenderness, and the muscles were found to be somewhat rigid. The kidney could not be felt. Urine, acid, sp. gr. 1016, contained albumen and some blood. Her condition appeared to be in other respects normal.

Whilst she was in the medical ward the attacks of pain were fairly frequent, but not severe; at one time it was noted that tenderness in the left hypochondriac and left lumbar regions was very marked; at another time she complained of a dull aching pain in her side, and generally her complaint was of pain in the left side. During this time the quantity of albumen varied, being at one time as much as a half. Some blood was nearly always present, but no renal casts were found.

Considering the frequent attacks of pain referred to the left side with the presence of blood in the urine, it was thought advisable to explore the left kidney for calculus; and on March 19 I operated by the lumbar route, and carefully examined the kidney and the renal pelvis, but without discovering anything abnormal. The wound in the lumbar region healed satisfactorily, and the patient went home on April 12, not having had any more pain. About a fortnight later she had a severe attack of pain, after which she was said to have passed blood in the urine, which was described as bright red in colour.

She was readmitted under the care of Dr. Hawkins on May 10, and between that date and June 4, when she was transferred to my care, had several attacks of pain of varying severity. The site of the pain was now more frequently about the umbilicus. Examination with the X rays did not show any calculus of kidney or of ureter. On June 7 she had an attack of pain which came on about 8 P.M., after she had been out of bed. She was "doubled up," had nausea and headache but no vomiting. On this occasion the pain was referred to the umbilicus, and extended outwards in all directions. Next day the urine contained about one eighth albumen and a trace of blood. The continued attacks of pain made it desirable to explore the abdominal cavity more thoroughly, and on the 15th June this was done through a median incision below the umbilicus. Nothing unusual could be felt about the kidneys or ureters, but when the under surface of the liver was explored a calculus could be most clearly felt impacted in the cystic duct. The lower opening was closed, and an incision carried through the outer fibres of the rectus made over the gall-bladder. The gall-bladder, which was slightly distended with mucus but not much thickened, was incised at the fundus, and attempts made to extract the stone with forceps and scoops, but without success. The stone was then held up and steadied, whilst an incision was

made directly over it. It was the size of a small marble, hard, rounded, and without facets. After cleansing of the opening in the duct, it was closed with interrupted Lembert's sutures of silk. The opening in the gall-bladder was fixed in the upper part of the wound, and a rubber tube placed in it for drainage. The remainder of this wound was closed in layers, like the exploratory incision. It was rather difficult to suture the opening made in the duct in this case, because the girl was very fat, there being at least two inches of fat on the wall of the abdomen. The sutures had to be applied at the bottom of a deep pit.

She left the hospital well on August 7, and has had no further attacks of pain. When she left the albumen had gone from the urine; this improvement has continued; it may have resulted from the prolonged rest in bed.

Case 2.—S. S., a single woman æt. 60, was admitted under the care of Dr. Sharkey towards the end of 1898, and transferred to my charge on December 20.

A week before admission she suddenly became sick and faint, vomiting freely. The vomit was only composed of froth. She has had three similar attacks since the previous September, during which (she says) the stomach was considerably distended.

On admission the abdomen is slightly distended, but moves well with respiration. There is no muscular rigidity. On palpation an elongated mass can be felt in the right hypochondriac region, which descends freely during inspiration. The lower border of this tumour, which is about four inches long and two inches broad, is clearly defined by a rounded edge. The internal and external borders can also be made out; the upper cannot be felt. The lower part of the tumour is hard, nodular, and extremely painful when touched. On percussion the note is impaired over the tumour, being continuous above with the liver dulness. Elsewhere the abdomen appears normal. Urine, sp. gr. 1030. No albumen, but a large quantity of urates.

On the 21st of December a longitudinal incision was made over this tumour, about three inches long, and the gall-bladder exposed. The fundus was opened and the interior examined for a stone; none was found in the gall-bladder, but search along the ducts showed the presence of one in the common duct near the duodenum. The gall-bladder had a very thick wall, being nearly a quarter of an inch in thickness. As it

was quite impossible to get at the stone from the interior of the gall-bladder, the duct was incised over it whilst it was steadied and held up to the surface, and the stone removed. Interrupted silk sutures were then passed after Lembert's method, and the incision in the duct closed. This was a difficult procedure on account of the distance of the opening from the surface. Afterwards the fundus of the gall-bladder was sutured to the peritoneum, and the wound in the abdominal wall closed in layers. A rubber drainage-tube was left in the gall-bladder.

There was nothing of moment in the after progress of the case. The opening gradually closed, and the patient left the hospital on the 28th of January, 1899, in good health.

One of the most interesting points in this case was the great hypertrophy of the wall of the gall-bladder. When felt through the abdominal parietes before operation it was so extremely hard that it gave the impression of being packed with large-sized gall-stones, on which it had contracted; this impression was made stronger by the feel of the surface of the gall-bladder, which was somewhat lobulated.

Case 3.—On February 25th of last year a married woman, æt. 49, was admitted under the care of Dr. Sharkey, and transferred to the surgical side under my charge on March 17, 1899.

She had suffered for eighteen years from severe abdominal pains, which began under the right costal margin, and extended round to the heart. The pain was accompanied by vomiting and general prostration. In the summer of 1898 she experienced her worst attack, and after this two gall-stones were passed, apparently about the size of hazel-nuts. During the last three weeks she had suffered from ten to twelve attacks of pain. Then three years ago she had an attack of pain followed by jaundice.

The patient was a fairly nourished woman, presenting no evidence of disease beyond slight enlargement of the liver, with some tenderness of the gall-bladder, the fundus of which could be felt. There was no jaundice.

At the operation on March 17 an incision was made downwards through the outer margin of the rectus, and the gall-bladder exposed. This was opened, and some three or four stones were removed from it; but some which could be felt in the cystic duct could not be extracted through this opening, so direct incision was made over them after enlarge-

ment of the abdominal wound, and more stones removed. There were others to be felt beyond these nearer the duodenum, but all attempts to extract them from the opening in the cystic duct failed, as they appeared to lie in a dilatation of the duct, and were packed closely together. They were about five in number, and marked by facets. Altogether about thirteen stones with a few fragments were removed, varying in size from that of a good-sized hazel-nut to small pellets. The incisions in the ducts were closed with a single row of fine silk Lembert sutures. The gall-bladder was drained after suture to the upper part of the wound. A second tube was passed deeply by the side of the ducts, and a gauze plug placed below it so as to completely isolate the ducts from the rest of the peritoneum.

The lower tube was removed on the 19th, and the gauze plug on the 20th.

At first there was very little discharge from the gall-bladder, but for a few days in the middle of April there was considerable escape of bile, enough to make the skin sore. This gradually diminished in quantity, and as the sinus was slow in healing the edges were pared and the opening sutured on May 5.

She left on May 17 with the wound soundly healed.

The first case is remarkable for the youth of the patient at the time of the operation, and for the curious variation in the pain and reference of its site to the left side of the abdomen. It was never typical of renal colic; but I think there are few surgeons who would not suspect renal rather than biliary calculus under the circumstances which obtained here, and few physicians who would not recommend an exploratory operation, especially when the repeated attacks which rendered her life miserable were associated with the presence of blood in the urine.

In many instances when the common duct is blocked by a stone the gall-bladder is shrunken, and too small to be brought to the wound and sutured there, so that it is necessary to provide for drainage in some other way than that adopted in these cases. I do not propose to discuss these methods of drainage, but think that if the gall-bladder can be brought to the surface it is advisable to secure it in the wound, and drain through it for the necessary time. An opening in the gall-bladder not only gives a chance for the detection and removal of stones, should any be in it, but by

emptying it of bile at an early stage in the operation there is much less risk of extravasation into the peritoneum; moreover the suturing of the incisions is more easily performed.

In a recent case of obstructive jaundice which I have operated on for Dr. Hawkins the distension of the gall-bladder diminished during the examination for the stone, and it seemed probable that it had passed into the duodenum during the search for it. A stone was removed from the gall-bladder. The jaundice passed off gradually after the operation, although no stone was found in the fæces.

XXIII.—*On a case in which a Gall-stone impacted in the Duodenal Orifice of the Common Bile-duct was extracted through an incision in the Bowel: recovery.* By J. Bland-Sutton. *Read May* 11, 1900.

A SPINSTER, 48 years of age, came under my care in the Chelsea Hospital for Women, April, 1899, on account of frequently recurring attacks of pain in the right hypochondrium, accompanied by vomiting and followed by jaundice and clay-coloured stools. Although the "attacks of pain" had troubled her over a period of three years, she had never, to her knowledge, passed a gall-stone.

On examination of the abdomen pressure over the gall-bladder provoked great pain, but no actual swelling could be detected. The skin was jaundiced, but not deeply, and the urine contained a trace of bile.

On May 1, I exposed the gall-bladder through an incision in the right linea semilunaris: it was shrunken, firmly adherent to the adjacent omentum, and free from concretions. The common duct was then traced to the duodenum. A firm body could be distinguished in the wall of the second part of the duodenum, and on carefully detaching the omentum I satisfied myself that a calculus had become impacted in the terminal segment of the duct. By careful squeezing I tried to push it into the bowel, but without effect. I then endeavoured to squeeze it up the common duct and return it to the gall-bladder: this proved equally a failure. Then by means of gauze dabs the operation area was isolated, and the duodenum opened through its anterior wall. The exposed bile papilla drawn carefully upwards by forceps could then be nicked with the scalpel, and the calculus squeezed out, followed by a jet of bile. The incision in the duodenum was then sutured in two layers,—a continuous silk suture for the mucous membrane, and interrupted silk sutures for the serous and muscular layers. In closing the incision in the abdominal wall I left a thin roll of gauze to act as a drain, the gauze reaching as low as the duodenum.

The wound healed, and so far as the abdomen is concerned all went well. Fourteen days later right suppurative parotitis,

due to staphylococcus infection, declared itself and delayed recovery. However, the patient left for a convalescent home six weeks after the operation, and in December, 1899, reported herself in good health.

The calculus, as large as a Barcelona nut, was, soon after its extraction, examined bacteriologically by Dr. Addinsell. The centre contained a small cavity occupied by fluid abounding in *Bacillus coli communis*, from which a pure culture was obtained.

It is well known that injuries to the abdominal viscera are not infrequently followed by infective parotitis, and in this instance I expected to find the *Bacillus coli* in the pus from the parotid gland; there was no inflammation or irritation around the abdominal wound, and this strange sequel of injury to the abdominal viscera remains quite obscure.

XXIV.—*Pneumothorax without Effusion in an apparently healthy man.* By F. Parkes Weber, M.D., F.R.C.P., Physician to the German Hospital, Dalston. *Read May* 11, 1900.

THE patient, G. B., a pianoforte fitter æt. 31, was admitted to the German Hospital on November 22, 1899, with well-marked signs of left pneumothorax. He had previously enjoyed average health. Never any serious illness except dropsy as a child. Never syphilis. Never hæmoptysis. Never any expectoration whatever. Never pleurisy or any lung disease; but has had a slight cough lately.

Family history.—His father is living, aged about seventy-eight, said to have an enlarged heart. His mother, aged sixty-eight, is living and healthy. He has had six brothers and one sister, and of these five are living and healthy. One brother died from an accident, and one at about thirteen years of age from dropsy. No history of consumption in the family.

On November 21 the patient, whilst doing his ordinary work at about 10 o'clock in the morning, was seized with severe pain (like a knife sticking into him) over the upper part of the front of the chest, chiefly to the left of the sternum. There had been no excitement or sudden or excessive exertion of any kind. His breath gradually became shorter, and the pain was increased on his attempting to walk. However, at about 10.30 A.M. he walked to the hospital with assistance, and was seen by the Resident Medical Officer, Dr. Scheffer. His respirations were 30—32 in the minute, and his pulse 65. The respiratory movements were equal on the two sides of the thorax. No cardiac impulse could be felt, and the cardiac dulness was displaced to the right of the sternum. The heart-sounds were natural. No epigastric pulsation. The percussion note was tympanitic over the lower left part of the thorax, and over the whole left half there was absence of respiratory sounds. Bell sound ("bruit d'airain," "coin-test") was present. The patient would not on that occasion stay in the hospital, but returned the following morning in a cab, and was admitted. The jolting of the cab made the pain in his chest worse.

When I saw him on November 23 the patient, a fairly

healthy-looking man, was resting quietly in bed, and did not appear distressed in any way. The pain in his chest had already practically gone. The signs of left pneumothorax were then quite typical, but there was no Hippocratic splash.

November 25, 1899.—The movements and vocal vibrations on the left side of the thorax are somewhat less than on the right side. Over the whole left half of the thorax the breath-sounds, voice sounds, and cough are all of amphoric character, and there is a distinct bell sound, but no metallic tinkling or splash. There is a small area of cardiac dulness to the right of the sternum. Respirations 20; pulse 76. The patient can sleep equally well on either side.

November 30.—The condition remains similar, but there is now no bell sound to be made out over a considerable area of the left side of the chest front, namely, over the upper and inner portion below the clavicle and to the left of the sternum. Pulse and respirations remain quiet, and the temperature has not been raised beyond 99·4° F. The treatment has chiefly consisted in rest in bed, a light diet, an aperient when required, and a belladonna plaster at one time to the left of the chest.

December 14.—Bell sound and metallic quality of the voice sounds can still be made out, at least over the lower left front of the chest.

December 21.—*Lungs.*—No distinct bell sound anywhere, but the percussion sound is more tympanitic, and the breath-sounds and vocal vibrations are much less distinct over the whole of the left side of the chest than over the right side. The most distinct cardiac dulness is still to the right of the sternum. There has never been any Hippocratic splash; and some tinkling,* which was observed near the stomach, probably proceeded from the interior of that organ. The

* According to Dr. de Havilland Hall (*Trans. Clin. Soc. Lond.*, 1887, vol. xx, p. 160), metallic tinkling occurs in cases of pneumothorax where there is no other evidence of fluid in the pleural cavity. The tinkling in such cases might perhaps sometimes be conducted from the stomach. Professor Clifford Allbutt, however, gives a different explanation for this sound in a case which he narrates in an editorial note in his *System of Medicine* (vol. v, 1898, p. 383). The patient was a healthy athletic young man about twenty years of age, in whom the pneumothorax was brought about by an attempt to bend the body backwards to touch the ground in a particular way with the hands. The air escaped into the pleural cavity with a succession of tinkles or clicks, which were audible in all parts of a large room. The sounds continued for two hours or more, and inspirations, at any rate at first, increased their number and loudness. Allbutt suggested that they were due to the bursting of a tiny bubble at each issue of air. The patient soon recovered from his pneumothorax, and remained well.

patient's general condition and his body-weight remain unaltered. He is still kept in bed. No fever. The urine when tested about this time showed nothing abnormal.

On the following two days the evening temperature was slightly raised, but did not quite reach 100° F. The cause of the slight fever was uncertain. The cardiac dulness remained for some time to the right of the sternum, but the patient otherwise seemed quite well, and in two or three weeks he was allowed to get up, at first for a short time, and afterwards for a longer time each day.

On the morning of January 22, 1900, after patient had been out of doors, I could make the following note:

Heart.—The apex-beat can be faintly felt about the normal situation, that is in the fifth intercostal space about a quarter of an inch internal to the left nipple line. The cardiac dulness commences above at the fourth rib, and extends outwards to the apex-beat; no cardiac dulness can now be made out to the right of the sternum. The heart-sounds are natural; there is no murmur.

Lungs.—The expansion of the chest is natural and equal on the two sides. The percussion note is good over both lungs, but possibly slightly more tympanitic over the left than over the right. The breath-sounds, voice sounds, and other signs are normal.

The liver dulness commences above at the sixth rib in the right nipple line. Neither the liver nor the spleen can be felt below the ribs.

The patient's general condition is excellent, but the pulse is rather frequent at present, possibly owing to excitement. There has never been any expectoration.

On January 25 the pulse was found to be abnormally frequent, and the patient was therefore kept quiet for some time.

On examination of the chest just before he left the hospital for the convalescent home, on February 1, no abnormal signs could be detected. Examination again on various occasions showed that there was a tendency to great frequency of pulse on exertion or excitement. Some adenoid vegetations in the naso-pharynx (which may have been the cause of a tendency for "colds" to *fly to his ears*) were removed about the commencement of May. The patient then appeared in his ordinary state of health, and there were no signs of any disease in the thorax.

Remarks.—Reckoning from the onset of the pneumothorax

to the return of the heart to its normal position, the duration of this case was about thirty-two days. Excepting quite at the commencement the subjective signs were very slight throughout, and the patient could hardly be said to look ill; neither did he suffer from bad appetite or lose in weight. In all probability the opening from the lung into the pleural cavity closed spontaneously very soon after pneumothorax developed, and there was never any indication for aspirating or letting out the effused air. There was never any sign of the pneumothorax being complicated by effusion of serum, blood, or pus, nor was there any pleuritic friction or pleurisy detected after the collapsed lung re-expanded. An interesting feature of the case was the very gradual disappearance of the abnormal physical signs (of pneumothorax). The disappearance of the bell sound ("bruit d'airain") from the upper front part of the chest afforded the first evidence of a partial return to the normal condition; and some weeks later on, when the lung had further re-expanded, no bell sound could be heard anywhere, though the breath-sounds and vocal vibrations were still much less distinct over the affected side of the chest, and the heart was still considerably displaced. The incorrect position of the cardiac dulness was the most persistent of the abnormal physical signs, and when the apex-beat could be felt in its natural position (thirty-two days after the sudden commencement of the symptoms) the pneumothorax was regarded as cured.

Quite a number of cases, in which apparently healthy or nearly healthy persons have suffered from temporary pneumothorax, have been described or collected and tabulated by Dr. S. West,* Dr. Thomas Whipham,† Dr. F. de Havilland Hall,‡ and others. Occasionally the pneumothorax recurs. Thus my father, Sir Hermann Weber, has told me of an otherwise healthy, stout man, about thirty-two years of age, who on two separate occasions (with about two years' interval between the two attacks) developed pneumothorax, recovery on each occasion taking place within a few weeks. Dr. J. F. Goodhart§ has recorded a remarkable case in which a young man, aged twenty-four, suffered from pneumothorax, first on the right side, and a year later on the left side. Upon both occasions the effused air seems to have been fairly rapidly

* *Transactions of the Clinical Society of London*, 1884, vol. xvii, p. 56.
† *Proceedings of the Medical Society of London*, 1886, vol. ix, p. 237.
‡ *Transactions of the Clinical Society of London*, 1887, vol. xx, p. 153.
§ Ibid., 1896, vol. xxix, p. 109.

reabsorbed without giving rise to any evidence of pleuritic disturbance. There was absence of any apparent adequate cause, but Dr. Goodhart supposes that the rupture of an emphysematous bleb on either side was the most plausible explanation of the occurrence.

It is practically impossible for any strain to give rise to pneumothorax when the lungs are really healthy. Dr. S. West,* after recording experiments as to the pressure which lungs from the post-mortem room can bear without rupture, comes to the conclusion that, in the absence of any disease of the lung, pneumothorax can only be produced in cases where the normal resistance of the lung is greatly reduced. Moreover, when rupture of the lung does occur, the result, he says, is usually subpleural, mediastinal, and cervical emphysema, and only very rarely pneumothorax. Thus, in the violent paroxysms of whooping-cough and the straining of parturition, subcutaneous emphysema is far more likely to occur than pneumothorax.

In many of the cases the onset of the symptoms, following a sudden strain of some sort, lends weight to the view that a slight tear in the visceral pleura at a spot where it happens to be bound by an adhesion to the parietal pleura may be the cause of the trouble. Thus Dr. I. Levison,† of Siegburg, gives the case of a young man, aged nineteen years and a half, who all at once on laughing violently experienced a sensation as if something in his chest had been torn, and felt a pain on the right side. He was found to have pneumothorax of the right side, the signs of which disappeared in about fourteen days. Dr. Levison regarded the pneumothorax in his case as probably due to the bursting of an emphysematous bulla. In my present case and in some others there was no history of any adequate strain preceding the onset of the symptoms, and the most likely explanation to account for the occurrence in these cases, and in most cases, is either that an emphysematous bulla gives way, or that a minute subserous tuberculous patch breaks down, and thus gives rise to a communication between the air-passages and the pleural cavity.

Some of the patients, like the one‡ whose history Dr. S.

* The "Bradshaw Lecture on Pneumothorax," *British Medical Journal*, 1887, vol. ii, p. 393.

† *Münchener medicinische Wochenschrift*, 1899, No. 41, p. 1341.

‡ Dr. S. West, in his "Bradshaw Lecture on Pneumothorax" (*Brit. Med. Journ.*, loc. cit.), says in regard to this case, "I have repeatedly examined the

West has given in the *Transactions of the Clinical Society* (1884, vol. xvii, p. 56), were probably the subjects of tuberculous pulmonary lesions; their previous or subsequent history suggests this, and Dr. West* has shown that pneumothorax occurring in a patient with even obvious signs of phthisis may be recovered from without any liquid pleuritic effusion taking place. Judging from the results of post-mortem examinations, it is certainly not necessary for patients with only small tuberculous lesions in their lungs to present any distinct signs or symptoms of tuberculosis. The remarkable fact pointed out by Dr. Whipham and Dr. de Havilland Hall, that the age of most of the patients is under thirty, may likewise, I think, be put forward in favour of a tuberculous lesion as the cause of the pneumothorax.

The breaking down of a minute subserous tuberculous lesion and the rupture of an emphysematous bulla (especially if the emphysematous bulla be attached by an adhesion to the parietal pleura) may probably both be regarded as giving rise to these cases of pneumothorax in apparently healthy persons, but since post-mortem evidence is practically wanting, nothing can be said as to the relative frequency of the two causes. When an unusual strain has preceded the onset of the symptoms, the rupture of a small tuberculous nodule affords as good an explanation for the occurrence of pneumothorax as the rupture of an emphysematous bulla. In my case there was no history of sudden exertion or strain, and making all allowance for the fact that a sudden exertion or strain is much more likely to occur in males than in females, I cannot understand why practically all these pneumothorax patients should be males, unless it be that subserous emphysematous bullæ are (like ordinary pulmonary emphysema) more common in males than in females, and that the bursting of such an emphysematous bulla into the pleural cavity is the cause of the pneumothorax in a large proportion of the cases in question.

chest with great care, and have always failed hitherto to obtain any definite signs of local lesion. The patient has, however, had three or four attacks of slight hæmoptysis, and I think the probabilities are in favour of a phthisical lesion. It is now four or five years since the patient had pneumothorax, and he continues in his usual health."

* "Bradshaw Lecture," loc. cit. The case is one of a phthisical girl, aged 19, who developed pneumothorax of the right side. There were no signs of any liquid in the pleural cavity. After a few days the air began slowly to disappear, and in about nine weeks the lungs had re-expanded, and the breathing was audible as before over the whole side. The tuberculous disease, however, advanced, and the patient died soon afterwards.

The only fatal case which appears to me to throw real light on this matter is that of a young man aged eighteen, described by Dr. Newton Pitt at this Society, February 9, 1900. The man in question suddenly developed acute pneumothorax with hæmothorax, and died in about twenty-four hours, doubtless mainly owing to the hæmorrhage into the pleural cavity. No perforations or injuries could be found after death, nor was there any trace of a tuberculous, inflammatory, or necrotic lesion in the lung, but there was an emphysematous bulla, about half an inch in size, torn open, situated near the apex of the lung; and attached to this was a ruptured adhesion, from which the blood might have come.

The duration of the signs in my case was not an unusual one. In the twenty-four cases (including three traumatic ones) collected by Dr. S. West, the majority, he says, made a rapid recovery; some were well in a few days, others in a few weeks; the longest took eleven weeks for complete recovery. In the table of ten cases brought forward by Dr. de Havilland Hall, with the exception of one that recovered in about eight days, all took from one to two months.

XXV.—*A case of Gastro-enterostomy followed by Pylorectomy: recovery.* By Arthur E. Barker. *Read May* 25, 1900.

WHEN an attempt is about to be made to excise the pyloric end of the stomach for carcinoma, most surgeons must experience, I think, considerable difficulty in deciding which of the numerous operative procedures to employ which have from time to time been devised. As some slight help to such a decision, I venture to present the following case to the Society, in which a procedure was adopted which has so far given the happiest results. The case has also some points of intrinsic interest quite apart from the result.

Pylorectomy.

Mrs. V., æt. 55, admitted University College Hospital on March 11, 1899. The patient had suffered at different times from attacks of pain in the right iliac fossa. The first occurred January, 1898, the last (about the fourth) six months ago. They were accompanied by slight vomiting and constipation, but no lump was ever noticed. No family history of malignant disease.

Seven weeks ago patient suffered considerable pain in the stomach, and was sick sometimes before and sometimes after meals. These symptoms had ceased for the last four weeks. The pain was mostly on the left side, and a lump was then noticed at times, but with only slight tenderness on pressure. Constipation was marked, and only relieved by cascara and enemata.

The patient was anæmic and thin. The tumour was about the size of a large walnut, its inner border lying at the *left* side of the *left* rectus muscle, and almost against the costal margin. It seemed remarkably superficial, and almost as if in the abdominal wall. It could be moved slightly from side to side, and also moved with respiration. There was no abnormality of liver, spleen, or kidney to be detected. It was variously diagnosed by those who saw it to be a growth in the abdominal wall, a growth in the splenic flexure of the colon, and an omental neoplasm. An exploratory operation was decided on.

Operation March 16, 1899. Eucaine analgesia, which answered remarkably well, was employed. Abdomen opened by a vertical incision at the outer border of the rectus, over the left side of the tumour, which was found to be the pylorus enlarged by malignant deposit. I at once performed a retro-colic gastro-enterostomy, using Carle's mode of securing Murphy's button without difficulty. The operation lasted one and a half hours, and was beyond doubt delayed by the fact that under local analgesia the operator has to manipulate more slowly to avoid the discomfort always present when the viscera are dragged upon, a sensation not obliterated by local analgesia of the abdominal wall. Very little pain was felt, but the patient became faint while the stomach was drawn forward for examination, and retched violently. This ceased on the stomach being released. There was no shock, and the patient had a cup of tea on returning to the ward. At 7 P.M. the pulse was 80 and of good tension, at 8.30 100, at 10 P.M. 80.

Recovery was only disturbed by some suppuration about the skin stitches, probably due to a small escape of stomach contents; otherwise the patient's general condition was surprisingly good, and she was soon taking abundance of food, beginning of course with very small quantities. There was no sickness. On the fourth day the stitches were removed, as there was suppuration under the skin around them. After this there is nothing to note but steady improvement in every way until the patient was taking average food and digesting well. The button was passed on the eighteenth day without trouble. The skin wound contracted, and soon became a superficial line of granulations.

At the patient's and her husband's request I agreed now to attempt the removal of the pyloric growth. As there was a new outlet for the stomach working well, it seemed probable that this could be effected rapidly and safely.

Second operation, April 20, 1899. The old scar, which was still granulating superficially, was first excised under chloroform, and then enlarged upwards. I first examined the posterior gastro-jejunostomy anastomosis, which was found to be perfectly satisfactory. Then after separating the omentum, great and small, by means of forceps and scissors, I clamped the stomach and duodenum, and excised the pyloric end of the stomach between. Unfortunately the clamp on the stomach was not tight enough, and the latter slipped from it, allowing some mucus and a little fluid to escape. This was rapidly wiped up, and the distal end of the stomach was closed by two

rows of continuous sutures, the first including all the coats and the second the serous and muscular, and folding the first row in on itself. The duodenum was similarly dealt with, and when all the protecting pieces of sterile gauze had been removed and the parts dried, the abdomen was closed by buried sutures, and the skin stitched separately.

After this operation, which was done under chloroform, the patient vomited considerably, and also on the next morning, in contrast to the first operation under local analgesia.

She was fed by nutrient enemata and hot water injections alternately every two hours, and one drachm of brandy with one drachm of milk were given every four hours by the mouth. This had to be stopped at 6 A.M. on account of vomiting. At 10 A.M. she was ordered half an ounce of peptones by mouth, but this had again to be discontinued on account of vomiting. Considerable pain was felt, and morphia was given hypodermically.

The next day feeding by mouth was commenced again, and was tolerated. Temperature 100·6°. Pulse 112.

From this on improvement was steady, in spite of the fact that the superficial part of the wound suppurated, probably owing to the fact that the first wound had been granulating at the time of the second operation. Of course it may have been due to the escape of a little of the contents of the stomach, owing to the slipping of the clamps. Be that as it may, the intra-abdominal condition was most satisfactory, and the chloroform vomiting having ceased, food was taken in increasing quantities, until solids were at last given, and the strength and weight were thereby improved. The wound was soon granulating kindly, and the bowels acting regularly. The patient left hospital on May 23, a month and two days after the pylorectomy, with a soundly healed wound, and weighing 6 st. 3 lbs. When seen on July 25, her weight had risen to 7 st. 4 lbs., a gain of 15 lbs. She then looked well, and the stomach and bowels were acting most satisfactorily.

Patient came to U. C. H. to-day (October 14, 1899) looking and feeling very well. She eats all ordinary food, and the bowels act more naturally. Weighed in a dressing gown her weight is 7 st. 6 lbs. Her husband says that before she became ill her weight was usually about 8 st. 2 lbs. in her ordinary clothes.

There are still two or three moist spots as though some deep stitches were going to work out. One has done so already. At this time there was no evidence of recurrence of

growth anywhere in the abdomen. The patient lived in comfort until the beginning of this year, when recurrence became evident, and she died exactly one year after the operation to a day.

The chief points of interest in this case appear to me to lie in the following facts.

In the first place the diagnosis was very difficult. The symptoms at first consisted of pain in the right iliac fossa with slight vomiting and constipation, which might have been set down to fæcal impaction. Only recently there had been pain in the epigastrium with more marked vomiting, but there was at no time much gastric dilatation. And when at last a tumour was found, it was in the splenic region, which, among all the many situations in which an enlarged pylorus is found, is one of the rarest. For my own part, I confess I did not even think of the pylorus, but thought the tumour was a growth in the splenic flexure of the colon, and prepared in every way to effect its removal. But when the true nature of the tumour was revealed, I at once did gastro-enterostomy with a view to removal of the pyloric end of the stomach if desired. This latter operation was urged upon me by both the husband and the patient herself, the result of the first operation having been so satisfactory. It was doubtless facilitated by the previous gastro-enterostomy, which only left an excision of the pyloric end of the stomach without anastomosis to be done. Besides this, the established outlet from the stomach allowed of early feeding by the mouth with very much diminished risk. Here the Murphy's button did good service in the retro-colic gastro-enterostomy, and there was no kinking of the bowel. It was secured in position by Carle's method, which has several advantages, as this and other cases have shown me.

Again, the use of local analgesia in place of general anæsthesia by chloroform in the first operation, seemed an advantage in this case. There was no vomiting after it as there was in the second operation under chloroform, preventing proper alimentation from the first.

Again, the division of the procedure into two stages diminished the risk of shock at all events, and perhaps of perforation. For it is easier to close a gastro-enterostomy opening securely than to unite the duodenum to the stomach satisfactorily. There is of course the drawback that the patient has to face two operations. This would in many cases be serious, and perhaps would determine me in other cases, as it has

determined many operators abroad, to combine the two measures in one procedure. But this is a question which invites discussion I venture to think.

Finally, it may be useful to others to know that in this case, as in several others, the posterior operation was found to be as easy as the anterior, and the conditions produced by it in regard to the examination of the stomach appeared to leave nothing to be desired. In all similar cases operated on since I have used the suture in preference to the button.

XXVI.—*A remarkable case of Obscure Septicæmia treated by Antistreptococcus Serum and Nuclein.*

By W. F. Victor Bonney, M.D. (introduced by Dr. Abercrombie). *Read May* 25, 1900.

I HAVE thought that the narration of the following case would prove of interest to this Society.

The patient, a young and previously healthy man of 33, had been taken ill thirty-eight days previously, so that this part of his case I can only detail to you from what was told me by himself, by those in attendance on him, and by the temperature charts that here I lay before you.

The onset was described as influenza, accompanied by an ulcerated throat, which was said to have been cauterised by the doctor he called in to attend him. He rapidly improved under treatment, and the temperature fell from 102·5° on the first day to 99° F. on the fifth day of the disease.

On the night of the fifth day he is said to have lain uncovered while asleep, and to have "caught cold."

On the sixth day the temperature was 103° and over.

On the eighth day he had a smart attack of conjunctivitis, lasting three days.

On the twelfth day the joints of the left leg began to swell, and on the succeeding day or two other joints; notably his shoulders and the right ankle followed suit.

The profuse sweats that were so characteristic a feature of the illness began about this time.

The condition was diagnosed by the medical man in attendance and by a consultant he called in as acute rheumatism.

On the eighteenth day the fever remitted somewhat, but the joints remained as before.

Soon after this he had great swelling of the glands of his neck (parotid?) and groin.

On the twenty-fifth day the fever took a higher grade; remaining pretty uniformly between 103° and 101° until the thirty-eighth day, when I saw him for the first time.

I may point out that the temperature chart up to this time is only a diurnal one, and therefore it is possible that he would have presented greater excursions of fever if the thermometer had been used every four hours.

During this thirty-eight days, constipation had been persistent, the bowels only reacting to daily enemata. The sweating had continued profusely since its onset.

When I saw him first his condition was as follows:

He was very wasted, sallow-faced, and sweating in a remarkable manner, so much so that his night-shirt had to be changed several times in an hour or two. The sweating had little or no influence on the temperature chart.

The left hip-joint was stiff, the left knee swollen, but quite moderately so, and the left ankle was slightly swollen. None of them were particularly tender.

The shoulder-joints were tender and stiff, the throat was no longer inflamed, and the neck and groin glands had subsided.

He was very deaf from the salicylates and quinine which had been freely administered to him, and was slightly delirious at night.

He had left femoral thrombosis, with a much swollen leg.

There was no urethral discharge, or any sign of swelling of the epididymes. Moreover the patient stoutly denied even the possibility of gonorrhœal infection. This I may mention he continued to do throughout his illness, even when very dangerously ill, though the subject was broached to him by many routes with a view to testing the veracity of his statements.

The abdomen was exceedingly distended and hyperresonant, but not tender. There was no enlargement of spleen or liver. The kidneys were not palpably tender or enlarged.

The heart presented very feeble sounds, particularly the first sound, which was valvular in character. There was slight enlargement of the cardiac area.

Examination could discover nothing amiss in the lungs. The urine contained a trace of albumen, was of low specific gravity, and there was nothing discoverable on microscopical examination.

The tongue was furred, there was no headache, and the eyes were normal by ophthalmoscopic examination.

Seeing he had had very large quantities of those drugs usually specific for rheumatic fever without result, and feeling sure that the condition was not at all events *pure* rheumatic fever, I omitted them, and contented myself with increasing his nourishment and exhibiting strychnine in the usual doses. A four-hourly chart was begun.

Though under this treatment his general condition improved—his delirium left him, and his deafness disappeared—

yet on the fortieth day he had a slight rigor, the temperature rising to 104·6°. I had him sponged with hot water.

On the forty-first and forty-second days he appeared better in himself, the extra nourishment and stimulants having a marked effect on his pulse. (He was taking 8 pints of milk, 1 pint of champagne, and 3 ounces of brandy in the twenty-four hours.)

On the forty-third day he had another very slight rigor, though the temperature rose to close on 105°.

On the forty-fourth day he had two severe rigors, the temperature rising to 106°. On this day Dr. Abercrombie kindly saw the patient with me. It was decided to use anti-streptococcus serum, which was begun that night. I took a hypodermic syringe full of blood from one of the superficial arm veins with the strictest asepsis to my friend Mr. A. G. Foulerton, who kindly examined it and reported it sterile.

It being impossible to exclude enteric fever, and the abdominal distension, continued constipation and femoral thrombosis lending some colour to such a theory, a Widal's reaction was sought for, but with negative result.

I may say that the diagnosis favoured at this stage of the illness was some malignant endocardial change. Against it was the absence of any cardiac murmurs or embolic signs. One could not ignore the diagnosis of rheumatic fever made by the eminent physician called in in the early stages of the case.

From the forty-fourth to the fifty-second day 220 c.c. of serum were given. The first 50 c.c. not giving rise to any improvement, a different serum was tried. This was made by Mr. Bokenham, and brought about a change much more marked than would be judged of from the temperature chart. Though the rigors did not immediately cease, yet they became much less severe, and the patient much less affected by them. Finally, although the rises of temperature occurred daily, yet they were unaccompanied by rigors. If the chart is referred to this is clearly shown. On the fifty-second day the temperature rose abruptly to 106·6°, but without rigor.

I may mention here that the temperature when bent on rising was quite uncontrollable by ordinary means. Neither sponging hot or cold, or the ice cradle, had any effect on it.

On the fifty-first day Dr. Abercrombie most kindly saw him again.

From the forty-fourth to the fifty-second day he had seven rigors. His condition in the earlier part of this period was critical, and the friends were told to prepare for the worst.

Under Mr. Bokenham's serum, however, a slow but obvious improvement took place, such as I have already detailed. The rigors became less severe, and delirium, which had been present during the onset of the stage of hyperpyrexia, diminished, although the temperature range had not yet fallen.

On the forty-eighth day, and onwards at intervals, pleuropericardial murmurs were heard, very typical. There were no new physical signs. The abdominal distension subsided with increased bowel action, induced by purgatives.

Stimulants were pushed to 10 ounces of brandy and 1 pint of champagne daily. Even when at his worst he took nourishment in a remarkable way, and it was not spared.

From the fifty-second to the fifty-sixth a marked and encouraging improvement took place. Rigors and hyperpyrexia ceased, and he mended rapidly.

On the fifty-sixth day the serum was reduced. From the fifty-seventh day, however, to the sixty-first the temperature steadily rose, and his condition generally deteriorated. On the sixty-third day, although the temperature had fallen somewhat, the friends were so anxious about him that Dr. Cayley joined Dr. Abercrombie and myself in consultation.

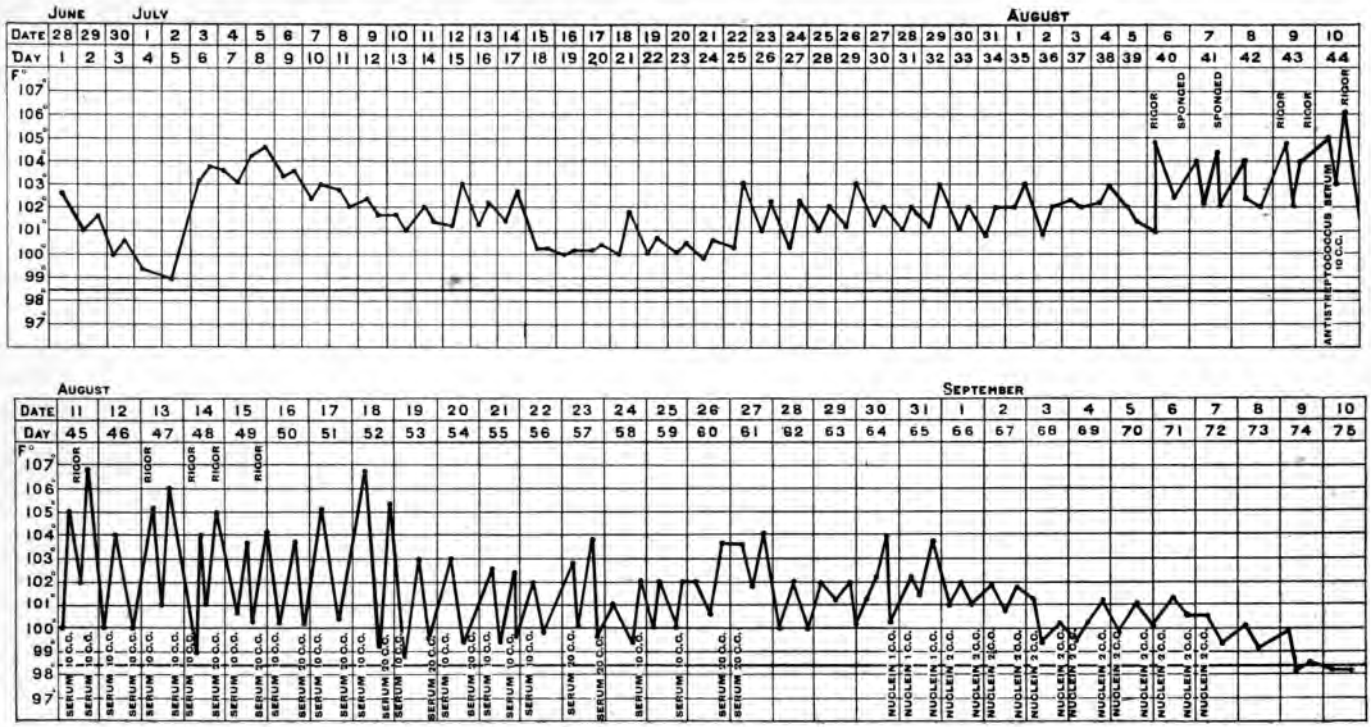
The diagnosis made now was Septicæmia, but from what source remained speculative. The serum, which seemed to have no beneficial effect on this recrudescence of fever, was abandoned; and at Dr. Cayley's suggestion nuclein, in the form of an alkaline solution, was injected hypodermically.

The nuclein was obtained from Messrs. Burroughs and Wellcome, who had previously made it for Sir R. Douglas Powell, to whose Lumleian Lectures for 1898 I am indebted for information concerning it. He quotes cases, and gives a temperature chart there.

I administered 3 c.c. of a 1 per cent. solution daily with excellent result, the fever after the first day falling steadily to normal, which it reached on the seventy-fourth day of the illness with no complication other than that on the sixty-eighth day well-marked pericardial sounds were heard, but they cleared up in a week. It never rose again, and the patient steadily convalesced.

On the same day that the nuclein was begun I attempted to aspirate the left knee-joint (the only articulation then swollen) for diagnostic purposes, but could obtain no fluid. The swelling had always been slight.

A day or so later Mr. Foulerton made a further blood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NUCLEIN 3 C.C.

examination for me, and reported the presence of one small colony of diplococci. By great misfortune he did not discover it to be a colony until he had placed the little opacity under the microscope. Consequently he was unable to make sub-cultures. He tells me, however, that he believes it was the gonococcus, but cannot of course be sure. A subsequent examination was reported sterile.

Looking at the whole case now, I believe it to have been one of Gonococcal Septicæmia, though it is easy to be wise after the event. Quite apart from the strong bacteriological suspicion, the manner of the joint infection—its persistence, and particularly the fleeting initial attack of conjunctivitis—which I find Dr. Berkeley Hill describes as a not infrequent forerunner of gonorrhœal rheumatism, are weighty points in its favour.

The loss of bacteriological confirmation is to be deplored; but in spite of this I have ventured to bring the case before this Society as of not small interest, because I believe most firmly that, but for the treatment, the man would surely have died.

If it be argued that the improvement be an accidental coincident with the injections, it is at least unlikely that such coincidence would occur with both series of injections.

The patient received 420 c.c. of antistreptococcus serum in forty-two injections. I believe larger quantities than this even have been given. All the injections were under the abdominal skin. No ill results of any kind were noted, except an urticarial condition towards the close. The skin became thickened, so that it would not pinch up easily.

May I be allowed to make one practical point? The needles of the serum syringes sold at the various instrument makers are too feeble and frail. Much less pain is given by using a stouter weapon, especially when, as in this case, the skin becomes thickened and cannot be pinched up.

It will be seen that 29½ c.c. of nuclein in 1 per cent. solution were given, either in the arms or legs, in sixteen injections; no ill effects were noted. I am indebted to Messrs. Burroughs and Wellcome for supplying me, free of charge, with this preparation.

I owe much to the kind assistance of Drs. Abercrombie, Cayley, and Foulerton; nor can I omit to mention the excellent nursing which so much determined the successful issue of the case.

XXVII.—*A case of Myxœdema supervening on Graves's Disease.* By W. Pasteur, M.D. *Read May* 25, 1900.

BEFORE reading the conclusion and *post-mortem* notes of the case of Mary Ann S., exhibited at a clinical evening in April, 1898 (cf. vol. xxxi, p. 305), I will recapitulate in a very few words the main features of the case at that time.

The patient, a widow æt. 65, was attacked twenty years before by Graves's disease. She was suffering from this disease in a typical form with tachycardia when first seen at the Middlesex Hospital in 1886. Under observation the enlarged thyroid dwindled to an "atrophic" state, but it is not stated whether this was accompanied by any change in the pulse rate.

When I first saw her, about 1893, there was well-marked permanent bradycardia, with an average rate of slightly over 40 beats per minute, slowing down to 30 or 35 during the severe paroxysms of cardiac dyspnœa, which formed the striking feature of her illness, and occasionally rising to 80 under excitement. There were signs of hypertrophy and dilatation of the left ventricle, with a rough systolic murmur in the aortic area, and a regular and fairly full pulse.

The demeanour and facial expression were placid and almost apathetic. She stated that her voice had altered in character, and she had become very sensitive to cold, and had more recently become rather clumsy with her fingers.

I believed at that time that she was the subject of degenerative lesions of the heart and aorta, and there was much speculation as to whether the bradycardia was related to the condition of the heart, or whether, taken in conjunction with the other symptoms just mentioned, it might not indicate the supervention of myxœdema. The latter view was the one most generally adopted by the members of the Society who examined the patient in 1898.

The subsequent history of the case can be given in a few words. She was readmitted to hospital on July 10, 1899, having enjoyed moderately good health in the interval, which, however, was broken by occasional attacks of cardiac distress lasting a few days. She had also frequently suffered from

attacks of giddiness and faintness which had occasioned several falls, in one of which she broke her wrist.

On admission there was marked cardiac dyspnœa, with some cyanosis and enlargement and pulsation of the external jugular veins. On examination the condition of the heart was practically the same as in April, 1898. The pulse was 36 per minute and regular; the lungs clear. Her chief complaint was of præcordial pain and faintness. Two days later the pulse-rate fell to 28, and throughout her stay in hospital varied between 28 and 36, touching 40 on one occasion only.

Under treatment she recovered sufficiently to go to the Convalescent Home at Clacton on August 8.

She was readmitted for the last time on January 4 last in a very critical condition. There was intense cyanosis with extreme dilatation of the superficial cervical veins, and marked signs of tricuspid regurgitation. Symptoms of heart failure had been present for about ten days. The pulse varied between 28 and 72 per minute during the twenty-four hours she survived her admission.

I am indebted to Dr. Campbell Thomson for the notes of the *post-mortem* examination, and the report on the microscopical appearances.

The patient died on January 5, 1900, and a *post-mortem* examination was made the following day.

There was well-marked proptosis; the body was well nourished.

The heart weighed 15 oz. There were several "milk spots" over the right ventricle, and excess of fat on the outside of the heart. The right ventricle was dilated, and was, together with the right auricle and great veins, distended with dark non-adherent blood clot.

The tricuspid orifice admitted nearly four fingers, the cusps of the valve were slightly thickened, but there was no hypertrophy of the right ventricle.

The pulmonary valves were normal, and there was no atheroma of the pulmonary artery.

The left ventricle was hypertrophied and dilated, the muscle of the ventricular wall was friable, but there was no fatty striation. The mitral orifice admitted nearly three fingers, and the anterior flap of the valve was slightly thickened.

The left auricle was not much dilated.

The aortic valves were competent to the water test; there was no narrowing of the orifice; the two anterior cusps were

DESCRIPTION OF PLATE XI.

Dr. Pasteur's case of Myxœdema.

A microphotograph illustrating the microscopical appearances of the thyroid gland described on page 191.

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slightly thickened and adherent to each other; there was also slight thickening at the base of the middle cusp. The coronary arteries were not much thickened.

The aorta was slightly dilated above the aortic valves; there were no atheromatous patches.

The thymus was not persistent.

The lungs were emphysematous and œdematous; there was a small quantity of clear fluid in each pleura.

The liver (50¼ oz.) was a little enlarged, smooth, and rather soft in consistence.

Gall-bladder and ducts normal.

The kidneys were somewhat granular, the capsule was slightly adherent; the cortex was not much diminished.

Suprarenals normal.

The spleen (1½ oz.) was very small and soft in consistence.

Uterus normal; ovaries fibrous and atrophied.

Stomach and intestines normal.

There were no obvious changes in the brain and meninges. The optic nerve was measured, and found to be half an inch longer than in another body of a woman of about same build, who died from thrombosis of the basilar artery. There was a good deal of fat in the orbit. There was nothing abnormal in larynx, trachea, and œsophagus. The thyroid gland was not abnormally small. Both lobes and isthmus were well marked. The two lobes were equal in size, and measured each 1½ inches in their long diameter. The right half of the isthmus was slightly fuller than the left half, but otherwise presented no abnormalities. On section the gland appeared normal in colour, but was of rather firmer consistence than normal.

Microscopical examination.—Thyroid: the appearance of sections varies considerably in different parts. In places the structure appears quite normal. The alveoli are well formed, and contain the usual quantity of colloid substance. In others there is an extensive round-celled infiltration which completely obliterates the alveoli, and in others an intermediate condition is present. At a rough guess about one half of the gland appears to be affected.

Sections of the heart muscle, liver, and kidneys were also examined, but beyond some excess of connective tissue, no abnormalities were found.

Remarks.—The belief that a true myxœdema may follow in the wake of Graves's disease appears to rest at present mainly on clinical evidence. I have not so far met with the

report of any case in which atrophic changes of the thyroid gland have been demonstrated *post mortem.* In this direction the case under consideration affords valuable confirmatory evidence.

In view of the condition of the thyroid gland revealed *post mortem,* it is interesting to note that the patient presented simultaneously some of the signs and symptoms both of Graves's disease and of myxœdema. For in association with a persistent exophthalmos, a smooth skin, tendency to perspiration, and occasional marked tremor of hand, we find an apathetic demeanour, extreme sensitiveness to cold, some clumsiness of the fingers, and alteration of the voice to a slight extent. I do not know whether slowing of the pulse to the extent observed is to be attributed to myxœdema or to some other condition. At any rate such a degree of bradycardia is most unusual in an uncomplicated case of myxœdema. The character and intensity of the attacks of cardiac distress—though peculiar as regards pulse rate—were much more suggestive of the heart storms of Graves's disease than of the syncopal tendency met with in myxœdema. It is also worthy of note that thyroid treatment proved of no avail in this case.

CLINICAL CASES

EXHIBITED.

*** Published in accordance with the regulation relating to the exhibition of patients at the meetings of the Society, viz. that each case shall be accompanied by a brief written description of the points it illustrates; such description to be published or not in the *Transactions* as the Council may direct.

I.—*Cerebellar* [?] *Cyst successfully treated by Trephining and Drainage.* By LEONARD GUTHRIE, M.D., and STANSFIELD COLLIER. *Exhibited October* 27, 1899.

A BOY æt. 9 was shown eighteen months after operation. Except for total blindness, due to optic neuritis, he was now in perfect health.

He was admitted to the Children's Hospital, Paddington Green, with symptoms of cerebellar tumour on February 15, 1898. His illness began about a year previously with attacks of vomiting and left-sided frontal headache. There was a history of a fall on the head followed by profuse epistaxis five years earlier, otherwise his health had been good.

The headaches at first occurred at weekly intervals, but in November, 1897, they happened nearly every day, and it was noticed that he "rambled" in walking. He had previously been a bright and lively child, fond of reading and of picture books, but lately had ceased to take interest in anything. He was very drowsy, yawned a great deal, and frequently slept for twelve hours at a time. His appetite failed, and he suffered from constipation.

On admission, well nourished; slight bluish congestion of skin, especially face and extremities. Cardiac sounds and pulse weak, otherwise normal. Respiration normal. There was no wasting or loss of muscular power in the extremities, but voluntary movements of the arms, especially the right, were somewhat ataxic. He walked with his feet wide apart, and his gait was reeling and uncertain—typically cerebellar. Knee-jerks equal and natural, other tendon reflexes normal. Examination of the fundi by Mr. Arnold Lawson revealed intense optic neuritis, with choked discs, swollen and very tortuous veins, and minute scattered hæmorrhages. The neuritis was evidently of long standing, but there were also signs of recent effusion. He could count fingers at a distance of about three metres with each eye. None of the ocular muscles were paralysed. There was no middle ear disease. Hearing and sensation were normal.

After admission his condition became progressively worse. The headaches occurred on most mornings, but occasionally he would be free from them for a day or two. The pain was invariably localised to the left frontal region, near the inner canthus. It was somewhat relieved by citrate of caffeine. Vomiting usually but not invariably accompanied the headache. There was frequently incontinence of urine and fæces by day and night.

Within a month of admission he gradually became unable to stand or walk without support, owing to tendency to fall backwards or from side to side. He became totally blind in the right eye within a fortnight, and in the left eye within a month of admission. Evidence of implication of vital medullary centres showed itself. His respiration became shallow, irregular, and sighing in character. The number of respirations fell to 13 to 15 per minute, and at intervals of fifty-five to sixty seconds a period of apnœa lasting ten to fifteen seconds occurred. The pulse was very weak at times, though not irregular, fast, or abnormally slow. The temperature, which for the first week had been slightly above normal (99° —99·8°), showed a tendency to become persistently subnormal (97°—98°). He was not particularly drowsy or somnolent, but much too quiet and contented in the intervals between the headaches.

The symptoms were obviously those of cerebellar disease, but there was little to aid in localising its site. The pain, as mentioned, was constantly referred to a small area near the left inner canthus. This, according to Purves Stewart, sug-

gested disease of the right cerebellum. But there was weakness of the lower half of right face, and the grasp of the right hand had become considerably weaker than the left, whilst the left knee-jerk, which had been equal to the right, almost entirely disappeared, the right remaining normally active. No local tenderness of the scalp was discovered.

On April 7, 1898, the left cerebellum was exposed in the usual manner. The dura mater bulged somewhat; it was thin and translucent. The convolutions were not flattened, and looked normal. Pulsation was almost if not quite absent. The only abnormality noticed about the dura mater was the presence of groups of minute yellowish-grey opacities, apparently in its substance. Nothing further was done except to close the wound.

On April 12 the right cerebellum was similarly exposed, and presented the same appearance as the left. The dura mater was opened on the right side next day. The cerebellum protruded through the incision; its substance looked normal. A trochar and cannula were introduced in a direction upwards, inwards, and forwards to the depth of about an inch and a half. On withdrawing the trochar about one ounce of deep yellow clear fluid spurted out with considerable force. The fluid was alkaline, became nearly solid with albumen on boiling, and did not reduce Fehling's solution; hence it was obviously not cerebro-spinal fluid.

A horse-hair drain was introduced through the sinus made by the trochar. The dura mater was sewn up, and the drain cut off close to its outer surface.

For a few days after the operation the scalp in the neighbourhood of the wounds was œdematous, and fluid collected beneath the sutures, but by April 23 the wounds were soundly healed and the stitches were removed.

The following notes of the results of operation were taken on May 19, 1898:

(1) The attacks of vomiting immediately ceased, and he has only once complained of slight headache.

(2) There has been no incontinence of urine nor of fæces since the operation.

(3) The embarrassment of respiration has disappeared. The pulse has increased in volume.

(4) The cyanosis is no longer apparent.

(5) His mental condition is much improved. He is cheerful, merry, and talkative. He eats and sleeps well, and has gained weight.

(6) Within the last week (a month after operation) he has been able to stand alone and walk with guidance; but he is still completely blind. The optic discs show signs of commencing atrophy.

Up to the present time, eighteen months after the operation, there has been no recurrence of the symptoms. It can only be a matter of regret that the operation was not performed a month earlier, as the boy's eyesight might thereby probably have been preserved.

The cyst might have been hæmorrhagic in origin, judging from its contents, but more probably it was a cystic glioma occupying the mid-lobe of the cerebellum.

Postscript.—September 6, 1900. The patient nearly two and a half years after operation still remains in good health. Occasionally he has slight headaches, but they are speedily relieved by small doses of calomel.

He is unsteady on his legs when unsupported, but this appears to be due to his blindness.

The area over the trephine holes pulsates freely. There is, however, no hernia cerebri.

The absence of recurrence of symptoms casts doubt on the diagnosis of cystic glioma. Possibly the cyst was not of the mid-lobe of the cerebellum after all, but a collection of fluid in the subarachnoid cistern, the vents of which had become blocked by inflammatory adhesions (posterior basic meningitis).

The curious yellowish grey opacities noticed in the dura mater at the time of operation were, perhaps, evidence of past inflammation.

II.—*A case of Actinomycosis.* By Geo. R. Turner. *Exhibited October* 27, 1899.

S. B., coachman æt. 42, was admitted under Mr. Turner's care into St. George's Hospital on April 28, 1899. Swelling commenced on the right side of cheek, Christmas, 1898, and slowly spread. He attended as an out-patient, and incisions were made beneath the jaw, "matter" being evacuated. The swelling, however, extended on to the forehead

DESCRIPTION OF PLATES XII AND XIII.

To illustrate Mr. Turner's case of Actinomycosis. A side and a full face view, before and after treatment.

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and right side of neck. On admission there was extensive brawny swelling over the right side of his face, the right eye being entirely closed, and on the right of the neck. The left eye was partially closed, and the brawniness extended over the left side of the forehead. There were several discharging sinuses, and the skin was much discoloured, red and purple in places, presenting an appearance of cellulitis with considerable congestion.

His general health had always been good, and he knew of no cause for his illness. The sinuses were laid open and scraped; but very little proper pus was evacuated. This proceeding was repeated in all some six times, whenever a new softening spot showed itself, and the patient was put on large doses of iodide of potassium, taking as much as forty-five grains a dose *ter die*. He bore the drug well, and manifested early and continued improvement. The brawniness commenced to leave the left side of his forehead, and improvement was marked at this situation, while the disease was extending elsewhere on the right. The photographs shown give a very good idea of his general appearance at various stages of the disease.

Report by Dr. Slater.

The pus and purulent material obtained by scraping the suppurating growth showed small yellowish specks of greater consistency than the rest of the material. These consisted of masses of mycelium, arranged in a radiate manner, and staining by Gram's method. The mycelium was of the size of that usually found in actinomycotic nodules, was dichotomously branching, but presented no clubs at the extremities of the mycelial filaments, though these were often somewhat swollen.

The same organism was found on subsequent occasions in the pus exuding from the growth.

Cultures were made on *agar*, and yielded a distinct but very scanty growth of minute dry, white colonies, in which the features of the original pus organism were well seen.

Inoculations on *glucose agar* failed.

Inoculations on *bouillon* yielded minute white, spherical granules. These consisted of filaments staining by Gram's method. The filaments stained very irregularly, presented swellings, and showed a marked tendency to break up into bacillary forms at the expense of the filaments.

On serum the growth was poor, occurred as milky white

pin-head colonies, and presented under the microscope the same features as the bouillon culture.

The organism is a streptothrix differing from the ordinary cultures of *Actinomyces bovis*, but resembling some of the many streptothrix organisms which can give rise to what is clinically actinomycosis.

III.—*A case of Ostitis Deformans.* By Geo. R. Turner. *Exhibited October* 27, 1899.

J. F., æt. 74, was admitted into St. George's Hospital September 15, complaining of swelling of the alveolar process of the upper jaw, pains in various joints, and swelling of the legs. The latter had been bad for six years. The enlargement of the jaws was of six months' (?) date, and had been treated by removal of all his remaining teeth at a provincial hospital. It was limited to the alveolar process, and was greater on the right than on the left side. There had been some increase in size of the head, marked by tightness of his hats and necessity to frequently change them. There is great thickening and curving forwards of the tibiæ and enlargement of the articular ends of both femur and tibia.

The upper extremities are normal, and he does not present signs of acromegaly. The urine is normal. He walks with a shuffling gait, with the chest and head bent forwards. Knee-jerks normal. Can stand in the dark. No venereal history. He has been treated by Pot. Iodid. and thinks he is better and that his head is smaller, as he can put his hat on more easily.

IV.—*A case of Excision of the Rectum for Cancer.* By J. Jackson Clarke. *Exhibited October* 27, 1899.

THE patient, a man æt. 56, had had symptoms of cancer for three months when he was admitted to the North-West London Hospital. On examination a growth was felt, its lower edge being about 2½ inches above the anus. Save at the back

it appeared to be free from attachments. After four days' preparation Kraske's operation was performed, and after closing the opening in the peritoneum about 5 inches of the rectum, and in connection with the rectum fatty tissue containing infected glands, was cleared away. The lower end of the upper segment was stitched in front to the anal portion, but for fear of sloughing the posterior part of the upper segment was not drawn down. The patient made an uneventful recovery, and the result appeared to the author to be better than that obtained by an ordinary colotomy, with the advantage that there was a fair chance that a cure might result.

February 12, 1900.—The present condition of this patient I consider to be very satisfactory. Since he was shown before the Clinical Society he has reported himself to me about once a fortnight. During this time, with the exception of a few days, he has been at his work and quite comfortable, having one motion daily, wearing a soft pad over the artificial anus, fixed simply by a T-bandage of "flannelette," a small double fold of lint being placed in the actual orifice. The latter does not contract, but is advantageously somewhat narrowed by the anatomical structures, sacro-sciatic ligaments, levator ani muscle, etc. A fortnight ago patient had constipation, accompanied by griping and abdominal distension, and followed by diarrhœa; the latter symptom being very uncomfortable, with the sphincter out of action. This condition was at once relieved by a dose of 3 grains of calomel at night, followed by a saline next morning. There was no evidence of any mechanical intestinal obstruction, and some marked nervous depression that accompanied and persisted after the attack made me suspect influenza, a suspicion that was confirmed by the good effects of salicylate of quinine, which he took for three days. There is no evidence of any enlargement of the liver. In view of the possibility of future attacks of diarrhœa I have recommended the patient to have a vulcanite plug made upon a vulcanite base, fashioned after plaster cast of the opening and its immediate vicinity, a plan I found to answer well in a former case of the same kind.

I should add that the skin around the orifice is free from any trace of irritation, and there is no suspicion of local recurrence in or about the parts now accessible to digital exploration.

V.—*Dislocation of the Right Hip-joint with Fracture of the Rim of the Acetabulum of over two months' standing reduced by operation.* By W. Watson Cheyne. *Exhibited October* 27, 1899.

W. G., æt. 60, stevedore, admitted to King's College Hospital on February 2, 1899.

About six weeks previously patient fell between two ships and was caught between the knee and the sacrum, the right thigh being bent at right angles to the body. He was taken to a hospital, and subsequently lay up at home.

When admitted patient was unable to stand on the right foot. When in the erect position the thigh was flexed and the foot everted. The right leg was an inch shorter than the left. When lying down flexion of the thigh on the abdomen could be easily carried out, also adduction and nearly complete extension: adduction was very limited. There was a good deal of pain on movement, and as the patient was very fat and muscular it was extremely difficult to make out the exact state of matters. On examination under an anæsthetic we came to the conclusion that it was a case of dislocation of the hip backwards, but occasional crepitus was elicited which we could not accurately explain. As attempts by manipulation under chloroform and also by pulleys failed to rectify the deformity, and as the patient was evidently hopelessly crippled, it was resolved to operate upon him in spite of his age.

On February 22, pulleys having been applied and the limb extended, an incision about 6 inches in length was made in the line of the anterior incision for excision of the hip-joint, and the wound deepened till the capsule was opened. On relaxing the pulleys the condition was made out to be dislocation of the head of the femur on to the dorsum ilii and fracture of the lower and back part of the rim of the acetabulum. The acetabulum itself was filled up with soft material. This was first cleared out and the fractured surfaces scraped, and then reapplying the pulleys all the parts were well stretched. Removing the pulleys the dislocation was now readily reduced by the ordinary manipulations. Care had to be taken that the head of the bone did not slip out again, which it was rather prone to do. When the head was reduced the fractured sur-

faces came into apposition of themselves. Limb put up somewhat rotated outwards, and abducted with long Liston splint and extension.

Wound healed by first intention. Splint left off on April 17, massage begun. Result seemed satisfactory.

Began to get up on 25th of April, and sent home on the 28th. He then got about on crutches, and could stand on the legs, which were of the same length.

On October 9, 1899, patient walked into hospital apparently quite well, and stated that he had resumed his work as a stevedore and was as well as ever he was. There was, however, still some slight restriction of all the movements, but not sufficient to interfere with his work.

VI.—*A case of Cystic Disease of the Kidneys.* By Sidney Phillips, M.D. *Exhibited October* 27, 1899.

A MAN æt. 40, whose health had always been good, except for occasional attacks of gout. In August, 1899, he fell over a cliff, and passed blood in the urine after this, but never before. On October 2, 1899, he experienced pain in the left flank, and blood appeared in the urine. He came into St. Mary's Hospital on October 9 with synovial effusion into the knee and ankle-joints, apparently gouty. There were found in the abdomen two tumours, one in each flank about the size of a cocoa-nut, irregularly ovoid in shape, with nodular surfaces. These tumours were fairly moveable, and could be pushed upwards just beneath the costal margin and downwards towards the pelvis; they were not painful or tender; there was no percussion resonance behind them. When admitted to hospital, the urine was blood-stained and contained $\frac{1}{20}$ albumen. Both blood and albumen had disappeared, and the urine was now clear and of specific gravity 1012. An average of 64 ounces of urine a day was passed. There never was any pus in the urine. There were no symptoms, and the patient was unaware that he had any tumours in the abdomen till they were discovered in the hospital. There was no cardiac alteration.

The symmetrical tumours, the absence of pain or other symptoms, rendered malignant disease improbable, and Dr. Phillips believed the case to be one of cystic disease of the kidneys.

There were no evidences of renal calculi or renal tubercle, and probably the cystic disease was of congenital origin, and had latterly taken on a rapid development.

It was not proposed to attempt any treatment, as the patient had probably little renal tissue left.

VII.—*Two cases illustrating Gastric Dilatation upwards and backwards, its relations to the Heart and Respiration, and its treatment.* By Wm. Ewart, M.D. *Exhibited October* 27, 1899.

PRELIMINARY REMARKS.—The two cases exhibited as a preliminary to a further communication illustrate a condition the symptoms of which are familiar to all observers, whilst its clinical anatomy has received little attention. They are both instances of chronic gaseous dilatation, as distinct from simple acute flatulent distension with its severe but temporary symptoms. In both groups, however, the mechanical result is often the same, viz. a marked rise of the left wing of the diaphragm, interfering with the freedom of respiration and especially with the heart, which may suffer from depression, arhythmia, discomfort, pain, or even angina. The stomach as a whole may be enlarged, but there need be no sacculation of the fundus, and its contents need not be permanently retarded in their normal progress.

The symptoms.—In those otherwise healthy upward dilatation is a common source of distress, but in advanced states of disease it constitutes a danger. Whilst a passing attack of flatulency is almost invariably recognised, this form of dilatation is often overlooked. In the absence of the physical signs and symptoms of sacculated fundus, the dilatation is apt to escape the notice of the physician, and the cases do not come under the surgeon's observation. The dilatation is, however, made obvious when, as in one of the cases exhibited, we succeed in bringing the vault of the stomach down to the

normal level, so that the enlarged viscus occupies the epigastric instead of the lower intra-thoracic region. Tympanitis, splash, fluctuation, etc., will then be obtained in the epigastrium.

The *special* symptoms are well known, viz. objective interference with respiration and with the heart's action; and subjective symptoms of breathlessness and exhaustion, of cardiac pain or distress, and of other discomforting sensations.

In the patients exhibited, both young (æt. 25 and 34), otherwise healthy, and free from cardiac murmurs, these symptoms have been sufficiently severe to partially or entirely unfit them for their work for considerable periods (two and a half years and six years respectively); they have become neurotic, and one of them an "air-swallower." In the latter (Case 1) the symptoms have been chiefly gastro-œsophageal and respiratory, viz. dyspnœa on exertion, apparently not cardiac, together with distressful deep-seated sensations at the lower sternal level.

In Case 2, although the heart and vessels appear to be normal, the cardiac symptoms have largely predominated. In addition to neuralgia, dyspepsia, and debility, which had lasted for six years, marked anginal attacks have supervened during the last six months with increasing severity. They are best relieved by remedies causing eructation of flatus.

In both patients the stomach was shown by the tympanitic note and by auscultatory percussion and auscultatory friction to reach a high level, shortening thereby the left pleura and limiting the movements of the left chest; and to rise behind the heart on its left side. As a result, resonance was substituted for part of the heart's dulness and of the left hepatic dulness, as shown by careful tracings taken from the surface markings.

The *ætiology* of upward and backward distension is varied, and will be discussed in a subsequent communication. In Case 2 the dilatation which extends into the epigastrium, as well as upwards, is of the chronic type common in dyspepsia due to bad teeth, and its mode of origin must have been largely "mechanical." In Case 1 the condition seems to have been induced suddenly by exhaustion from excessive physical exertion without sufficient food, and may therefore be regarded as neuroparalytic.

Treatment by operation does not appear to be needed for these cases. There is no obstruction to the passage of food; it is the gas which does not pass. Indeed, most of them are

instances of simple gastromegaly. Nevertheless, in some of the cases the upward dilatation tends to be complicated with sacculation of the fundus, and these may ultimately call for surgical relief.

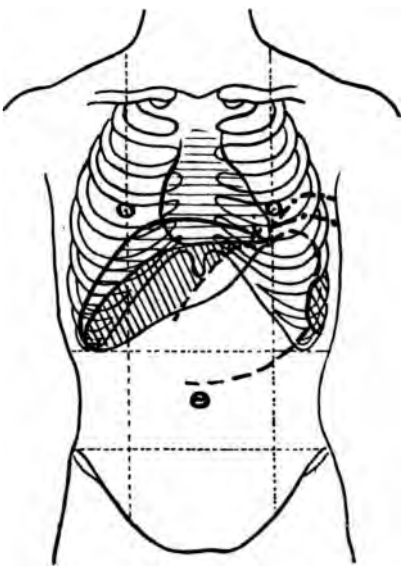
The rational indications which may be hinted at are:—(1) To treat the dyspeptic flatulence by medicine, hygiene, and diet. (2) If possible to keep the stomach from being over-distended; with this object lavage may have to be used systematically. (3) Treatment must also be directed to the faulty position and function of the diaphragm. The left wing of the diaphragm has to be brought down. This was successfully accomplished in Case 1 by suitable respiratory exercises, calculated to throw the labour of respiration chiefly upon the left side of the thorax, and to preclude the right side of the chest and the right wing of the diaphragm by posture from taking their full share in it. (4) A further indication, partly attainable by exercises addressing themselves to the abdominal muscles, is to strengthen the abdominal parietes, and to endeavour to reduce the capacity of the stomach and to obviate its sacculation after it has been brought down to a lower level by the descent of the diaphragm. The stomach should receive not only the lateral support of the diaphragm from above, but also that of the abdominal muscles from below; and a necessary adjunct is a hypogastric abdominal belt to be worn habitually, but particularly during physical exertion. The ballooning of the stomach will then run less risk of being converted into a sacculation. Case 2 has only recently come under treatment, and presents the original condition unrelieved. In Case 1 the tracings show the difference in level which has been obtained by systematic respiratory exercises bringing into action the left wing of the diaphragm, which has now resumed its proper relations, and has subsided from the level of the nipple to the normal level of Traube's area. Coincidently with this change the patient has partly regained his capacity for muscular efforts, the performance of which in his previous state he had declared to be an absolute impossibility.

Case 1.—A man, S., æt. 25, a cycle maker, previously healthy, had been incapacitated from work for two and a half years, since an exhausting bicycle ride followed by a wetting, owing to constant discomfort seated at the lower sternal level, aggravated by food, and complicated on exertion by a distressful dragging and cramping sensation over the præ-

cordium, radiating upwards and outwards towards the left axilla. He has had no fainting, giddiness, palpitation, irregularity or stoppage of the heart's action, nor any other severe symptom.

The deep-seated sensation at the epigastrium is eased for a moment by eructation, and he has become a "wind gulper."

FIG. 7.



The interrupted line shows the present tympanitic area encroaching upon the hepatic dulness. The interrupted lines with dots show the original upper level of the vault of the stomach, and an intermediate level found at a subsequent examination. (From tracings exhibited.)

Diet has no influence upon the symptoms, and there is never any severe indigestion. All kinds of food are welcome, but the same symptoms of flatulency occur after all of them.

On admission into St. George's Hospital on August 24, 1899, he appeared healthy, but pale and nervous. All the organs were apparently healthy. The abdomen was spare, and gave no signs of gastric dilatation. He had been suspected of some chronic mediastinal disease, but no evidence could be obtained of this either anteriorly or at the back.

The stomach resonance rose high into the thorax, upwards

as far as the lower left nipple level, and backwards nearly as far as the scapular line.

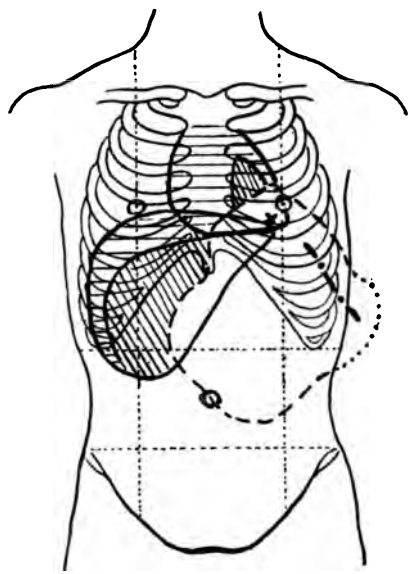
The treatment was directed to the general health, to the stomach and digestion, to the heart, and to the diaphragm. Purgatives, carminatives, and antispasmodics made no appreciable difference in his condition. The chief results were obtained from mechanical treatment by respiratory, thoracic, and abdominal exercises tending to cause a descent of the left wing of the diaphragm. Nauheim baths were used as an adjunct to hygienic measures.

Whilst the patient improved in weight, in muscular strength, and in capacity for exertion, he did not lose his deep-seated sensations, which seemed to be localised at the cardia, nor his general feeling of internal discomfort. The improvement in the breath and in muscular energy is probably connected with the reduction of the upward and backward dilatation of the stomach. The tympanitic resonance has been gradually but quickly brought down to the normal upper level indicated by the surface markings; but as a result of this change moderate dilatation can now be made out in the epigastrium by palpation and by percussion. When the stomach tube was passed no obstruction could be felt either in the œsophagus or at the cardia. But it is significant that its passage, in the patient's own words, "touched the spot," and afforded decided though temporary relief. Subsequently the stomach was washed out, also with relief; but by degrees the daily repetition of that operation ceased to bring the same alleviation, and there being no evidence of faulty digestion, the practice was given up. Three and a half pints of fluid could be injected into the stomach, but at that stage the patient declared that he could hold no more. The dilatation may, therefore, be regarded as one of moderate extent, and it is probably a uniform dilatation without any gastric deformity.

Case 2.—G. S., æt. 34, a timber labourer six feet in height and powerfully built, was admitted into St. George's Hospital on October 23, 1899. His aspect was healthy and free from pallor, but neurasthenic. For six years he had been in indifferent health, constantly feeling unfit for work; and for years he has been useless as a workman. He denies any syphilis or alcoholic excess, and the family history is healthy. His early symptoms were those of dyspepsia secondary to bad teeth. These are now in a lamentable state, and several of the stumps exceedingly tender. His neuralgic pains and

general symptoms have been much worse during the last six months, and have become associated with definite anginal attacks of cramp and suffocation at the heart, and with a feeling of fulness in the chest which seems to extend upwards

FIG. 8.



The tympanitic gastric area encroaching upon the areas of absolute dulness of the liver and heart is shown in interrupted line (dotted beyond the mid-axillary line). The interrupted line with dots indicates the lower level found at a later date. (From tracings exhibited.)
N.B.—The left chest was bulged outwards, and the xiphoid displaced to the left.

as far as the left axilla, with pain and numbness down the left arm or both arms, and also up the left side, and sometimes also the right side of the neck to the sterno-mastoid region immediately behind the auricle. There is also a feeling of stoppage of the heart. Almost every night since his admission he has had more or less severe seizures, usually relieved by amyl nitrite or by ether pearls. He describes the attack of pain as beginning in the epigastrium; a throbbing pain sets in next at the occiput and soon after in the jaw, and finally the præcordial distress and the radiating pain in the arm and neck attain their full development. There has also

been occasional pain in the right flank, thigh, and leg, but this has not been associated with the attacks.*

The urine (sp. gr. 1010, slightly alkaline) contained no albumen. The lungs were normal. The apex-beat was felt in the fourth space, an inch internal to the mammillary line. The apical first sound was of rather grating character, but not otherwise abnormal.

From the first the case struck me as being probably one of dilatation of the stomach upwards and backwards, and this on examination proved to be correct. But the stomach was also distended downwards over a considerable surface of the epigastrium as well as backwards. It extended upwards to an inch and a half above the mammilla, towards the right (at the lower sternal level) to an inch and a half beyond the middle line posteriorly, and upwards as far as the posterior axillary line; and the lower boundary reached a level two and a quarter inches below the navel.

The extent to which the stomach rose behind the apex of the heart is well shown in the tracing of October 24. A tympanitic resonance was found to take the place of the lower part of the absolute cardiac dulness, and over the remainder of the latter the dulness was lessened in degree; nay, in some postures almost the entire dulness as far as the sternum was replaced by resonance. A clear distinction could be made by auscultatory percussion between the tympanitic note transmitted from the stomach and that distantly propagated by conduction through the media of the heart. At the back, auscultatory percussion also verified the extension originally traced out with the pleximeter.

In spite of the low level attained by the fundus, the absence of vomiting, and of any evidence of difficulty in the transit of the ingesta, seems to identify the case as one of gastromegaly with little if any sacculation. The causation is probably to be traced to habitual gaseous distension from the intra-gastric fermentation of ill-masticated food.

The cardiac seizures are doubtless partly reflex and partly dependent upon general and cardiac asthenia, but they would seem to be immediately occasioned by the direct mechanical

* Since the date of this report the patient ceased to have anginal seizures after the tender stumps had been removed; but he has suffered much, both in the night and after walking a few steps, from severe pain striking through the right hypochondrium, and radiating down the thigh and leg and up the spine into the occiput. The pain is relieved by lying down. It coincides with flatulent distension of the ascending colon, and is eased by the escape of flatus.

interference of the gastric distension with the perfect freedom of the heart.

In cases of this kind the firm and relatively unyielding pressure exerted upon the apex from below and from behind by an inflated stomach stretching the tough membrane of the diaphragm over it, takes the place of the soft contact and of the oscillating support of the elastic lung with its negative pressure, and this may prove too much for a damaged heart, for a cardiac muscle weakened by mal-nutrition, or for cardiac nerves worn by constant pain.

VIII.—*A case of Scleroderma and Sclerosis of the Muscles with recurrent attacks of local Syncope.* By W. P. Herringham, M.D. *Exhibited October* 27, 1899.

EDWARD C., æt. 30, a worker in tobacco. Has had pains in hands, forearms, shoulders, and knees since Christmas last, which he calls rheumatism. Since April the left hand, and more recently the right hand also, have been subject to attacks of stiffness, swelling, and deadness. The ears get cold at the same time.

Was seen on June 27. Both hands dead white and stone cold. The radial pulse natural.

On admission to the ward the condition had passed off, and he has had no similar attack since then, having been in bed nearly all the time.

He is pale and poorly nourished, with livid lips. About the malar bones, on the wrists and hands, in the loins, and down the lower extremities the skin is hard and tight. He cannot bend the trunk at right angles to the legs, nor move the legs freely. The wrists are half bent, and the fingers almost straight and nearly immoveable. The hands much resemble those of rheumatoid arthritis in an early stage. The fingers are smooth and tapering, and the joints, though not really enlarged, seem so from the tight and shrunk condition of the phalanges between them. The tendons of the fingers grate or creak in the sheaths on passive movement, and so do those of the wrists. This might easily be mistaken for articular grating. The muscles of the forearms and those of the thighs

and legs are hard and contracted, so that he can move the hands and legs but little. On passive movement there is great rigidity. The knees can be extended, but can be bent only to a small extent. As they are bent the quadriceps can be felt grating in its sheath. The ankles are similarly affected. The deep reflexes are much exaggerated at the knee and elbow, and the contraction after the radius or the tibia has been hit is clonic. The muscles seem very irritable to this stimulus.

He has little pain, yet looks as if he had much.

[The condition of the muscles is a fibrosis, with disappearance of muscular fibre. It has been well described from post-mortem appearances by Thibierge and Vandervelde. The whole disease is usually thought to be due to some central nervous affection, but nothing has yet been found to prove it. A full bibliography is given by Lewin and Heller, *Charité Annalen*, 1899, vol. xix, p. 763.]

Postscriptum.—After several weeks' stay in St. Bartholomew's without improvement, he went to the hospital at Bath for hydropathic treatment. While there he died suddenly. An autopsy was refused.

IX.—*A case of Recurrent Carcinoma of the Breast treated by oöphorectomy and the administration of thyroid extract.* By Frederic Eve. *Exhibited October* 27, 1899.

MISS B., æt. 40, consulted me in October, 1895. Three years before she noticed a lump in the right breast. At the outer edge of the gland was a superficial cancerous ulcer, covered with a scab. Its base was deeply retracted, and not adherent to the subjacent structures. There was little apparent induration of the surrounding tissues. The nipple, which was indrawn, was situated about one inch from the ulcer. No enlargement of the axillary glands could be felt, but there was a suspicion of glandular enlargement above the clavicle. Her mother died, aged sixty-three, of an abdominal tumour, probably carcinoma of the ovary.

On November 11, 1895, the breast was completely removed,

the axilla cleared out, and a slice of the pectoral muscle excised. Primary union.

Three years later, on July 9, 1898, I found a small carcinomatous nodule of the skin above the inner third of the cicatrix, and there was a puckered thickening of its centre. Two distinctly enlarged glands could be felt above the clavicle. She had not lost weight. A few days later oöphorectomy was performed. Soon after she began to take thyroid extract, gr. xv per diem.

October 20, 1898, the cutaneous nodule and the thickening of the cicatrix had almost disappeared. The enlargement of the supra-clavicular glands had also much diminished. Two fresh cutaneous nodules, however, had appeared above the inner half of the cicatrix.

December 10, 1898, no change.

March 6, 1899, the cutaneous nodules and supra-clavicular glands had completely disappeared. Her general health was good.

June 21, 1899, three subcutaneous nodules had made their appearance above and below the inner half of the cicatrix. The glands above the clavicle were just palpable, and there was a distinctly enlarged gland at the anterior edge of the sterno-mastoid about the middle of the neck. She had been taking thyroid extract ever since the ovaries were removed.

About a fortnight later she was admitted to the London Hospital, and the cicatrix together with an extensive area of the skin was removed. The exposed surface was covered with skin grafts by Thiersch's method. The enlarged jugular and supra-clavicular glands were removed, a portion of the clavicle being taken away to facilitate this.

Remarks.—It may be noted that the carcinoma was of a subacute variety, it having existed for three years before the first operation. The patient's age was forty-three when oöphorectomy was performed. She was menstruating regularly at that time, but the catamenia ceased immediately after the operation.

The beneficial effect of this operation was most striking, and at one time it appeared as if a cure had been effected; but the subsequent recurrence of the trouble indicates that the morbid growth, although it had retrogressed, had only been rendered for a period latent.

X.—*A case of Severe Comminuted Fracture of the outer third of the Clavicle, treated by a new splint.* By C. E. F. Mouat-Biggs. *Exhibited October* 27, 1899.

A MAN of 29, having sustained the above fracture, was treated by a splint which I have had in use for fifteen years. I would claim that the amount of callus and deformity are less than by any other method.

The method of application is best seen from the accompanying photographs.

The splint secures greater fixation than any other method; is cool and comfortable; can be used for either side, and is invaluable in dislocation of the shoulder and clavicle.

It will be noticed that the axillary pad and the elbow support being in one, there is less tendency for the bandages to slip. It may be added that the splint is inexpensive, and that ordinary bandage may be used where webbing is not available.

XI.—*A case of complete Subperiosteal Resection of the Os Calcis: recovery, with reproduction of the bone.* By J. Hutchinson, Jun. *Exhibited January* 12, 1900.

REMOVAL of the os calcis in whole or part is usually undertaken for tubercular disease; and whilst of course many cases recover with a useful foot after erasion of a considerable tubercular focus in the bone, if the whole of it has to be removed the ultimate result is, I believe, usually unsatisfactory, and perhaps even inferior to that of an amputation. The following case proves that in the rarer examples of septic necrosis of the whole bone subperiosteal excision may be followed by most satisfactory re-formation of the heel.

The patient was a lad æt. 15, employed as a barman, who was admitted into the London Hospital on February 8, 1899.

DESCRIPTION OF PLATE XIV.

To illustrate Dr. Mouat-Biggs's Splint for Fracture of the Clavicle, described on page 212.

Adlard & Son, Imp.

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DESCRIPTION OF PLATE XV.

A skiagram showing the reconstruction of bone in Mr. Hutchinson's case of Subperiosteal Resection of the Os Calcis.

Adlard & Son, Imp.

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DESCRIPTION OF PLATE XVI.

To illustrate the appearance of the foot in Mr. Hutchinson's case of Subperiosteal Resection of the Os Calcis.

Allard & Son, Imp.

Mrqu

His right foot had been swollen and painful for five weeks, the inflammatory swelling extended from above the ankle to the toes, pus was evidently present, and shortly after his admission it was evacuated from around the heel by two incisions. The os calcis was then found to be bare. His temperature continued to be high (from 100° to 105° at night) in spite of further incisions and drainage. It became evident that the whole os calcis was undergoing necrosis, though the cause of this could not be exactly ascertained; he had had typhoid fever a considerable time previously; there was no reason to suspect a tubercular origin. In the extreme pain and high temperature and free suppuration the process resembled acute necrosis, such as attacks the long bone of young adults, due to staphylococcus infection. The patient continued very ill until March 4, when the temperature dropped, and on March 9 I operated, enlarging the sinuses on the outer side of the foot and saving the thickened periosteum, which was already forming new bone. The entire os calcis with the exception of the tip of the sustentaculum tali was removed in one piece; care was taken to leave the insertion of the tendo Achillis in its continuity with the periosteum of the under surface of the bone.

Section showed the interior of the bone to be riddled with pus. The large cavity thus left in the foot was packed with iodoform gauze, and the foot kept at right angles on a splint. He left the hospital on the 26th of April, then having one discharging sinus; in November he was readmitted on account of an attack of mild cellulitis, and through the kindness of Mr. Moullin he was transferred again to my care. The accompanying figure, taken seven months after the removal of the os calcis at a time when he could walk fairly well, bearing his weight on the foot, shows that the bone has been largely reproduced, sufficiently so to form a well-marked heel. At the same time it would be incorrect to say that the new os calcis is nearly the size of the old one, for careful examination of the skiagraph proves that it cannot be much more than half the normal size.

At the time of the operation the articular surfaces for the astragalus and cuboid were wholly destroyed, and the new bone has of course anchylosed with both these. Further, the head of the astragalus has completely united with the scaphoid.

Looking at the patient's age it is probable that the new os calcis will considerably increase in size, and the projection

of the heel is already quite sufficient to give him good leverage for the calf muscles and a firm support in walking.

In 1877 my late colleague, Mr. McCarthy, had under his care a similar case to the one just narrated,—similar, that is, in the fact that the whole os calcis was the seat of primary osteomyelitis. The patient was a boy æt. 2, and the disease was supposed to start as an acute epiphysitis; the ankle-joint, however, was invaded within two or three days, and it is therefore probable that the process was a general periostitis and osteitis from the first. It will be remembered that the true epiphysis of the heel does not begin to ossify until from seven to ten years after birth. In spite of incisions and drainage this patient died with pyæmic deposits in the kidneys, liver, and spleen.

This is the only similar case occurring at the London Hospital during the last ten years, and I have been unable to find any example of complete excision of the bone performed at the hospital during this time besides my own case. There are, of course, many examples recorded in medical works, and in some of them the almost perfect reproduction of the os calcis is emphasised.

XII.—*A case of Congenital Dislocation of Right Hip, with skiagraph.* By J. R. Lunn. *Exhibited January 12, 1900.*

ALICE M., æt. 5 years, was admitted with "eczema of the ears" on August 30, 1899. It was noticed that she did not walk well, and that there appeared to be some atrophy of the femoral muscles on the right side. The only history that could be extracted from the mother was that the child was born with a "shrivelled leg," and had consequently always limped.

The "impetigo contagiosum" soon healed under appropriate treatment.

About the middle of November the patient was radiographed, and copies of the condition examined.

The family history is not obtainable, and the question of

DESCRIPTION OF PLATE XVII

A skiagraph to illustrate Mr. Lunn's case of Congenital Dislocation of the Right Hip.

Avan & Son, Imp.

Mylar

heredity cannot thus be determined. The dislocation is unilateral and right-sided.

Symptoms.—Patient stands with the right foot markedly everted. Her gait is "waddling," and not so marked during running. An appreciable amount of lordosis is present, mainly from the curving in lower dorsal and upper lumbar vertebræ. The shoulders are well thrown back, and the normal line of gravity is thus behind the usual vertical axis. The hands reach low down at the side. The muscles are not much smaller on the affected side, and circumferential measure shows ½ inch in difference. The pelvis is, comparatively, somewhat broad. The right great trochanter is more prominent than the left, and nearer the iliac crest. The right tuber ischii is more prominent than the left, and not so covered as the left. As regards movements, abduction on the right side is limited, though a fair amount is obtainable; other movements are good. Lordosis disappears in the recumbent position.

Measurements.—The upper border of right trochanter is above Nélaton's line. From ant. sup. spine to right internal malleolus 19½ inches; from ant. sup. spine to left internal malleolus 20⅛ inches; from ant. sup. spine to right external malleolus 18¾ inches; from ant. sup. spine to left external malleolus 19¾ inches.

There is a limp on affected side, and the shoulder is lower. No appreciable wasting of gluteal muscles. No loss of gluteal fold. Traction causes disappearance of shortening, and makes ¾ inch difference. The general health and condition of the child are both good; no other abnormalities are present.

From the skiagram the following facts are obtained:—The dislocation appears to be upwards and slightly backwards. The cotyloid cavity is present, but shallow; the head of the bone is indented where it rests on the rim of the acetabulum, and the latter "can scarcely be described as misshapen," or missing a posterior rim, for on this the head appears to be resting. The shortening is due to mal-development and altered direction of the neck of the bone. The head, neck, and shaft of the bone are slightly atrophied, and are not so fully developed as on the sound side. The various epiphyses are very well shown in the copy.

XIII.—*A case of Paroxysmal Hæmoglobinuria.* By JOHN R. LUNN. *Exhibited January* 12, 1900.

ADELAIDE L., æt. 6, was in the infirmary recently (3rd to 28th October), being admitted for hæmaturia. Although the urine was frequently examined no blood was found. Patient was again admitted on November 28 for hæmaturia. Her urine has been examined on a great many occasions, no reaction for blood being obtainable until 9th December; no blood-cells were visible under the microscope. The urine is often very highly coloured, especially if patient has been exposed to the cold; but a positive guaiac and ether test is seldom obtainable.

On December 3 patient's sister called, when the following facts were communicated:—Patient has been in Middlesex Hospital seven times for hæmaturia; on each occasion she remained about three weeks. Six months ago patient went to the Great Ormond Hospital for six weeks. At each hospital a diagnosis of hæmaturia was made; the cause of this condition was, however, not ascertained. When patient is exposed to cold weather her water becomes highly coloured (sometimes as deeply coloured as port wine). When patient is up and active she experiences pain in the abdomen, chiefly on the right side; but also on the left. The pain often passes round to the back. During these painful attacks patient vomits (since admission she has had no pain, and has had not vomited), feels cold but does not sweat. The extremities particularly are very cold during the pain. Temperature 94·2°; pulse 92. Patient has not had scarlet fever. As long as patient remains in bed and keeps warm her urine is clear. Patient has never been known to pass any calculi *per urethram*, but she is said to have once had gravel. Patient looks pale and anæmic. No anasarca of limbs. On palpation of the abdomen no morbid renal affection can be determined.

I have seen her sister, and she is evidently suffering from syphilis, choroiditis, and interstitial keratitis.

The red cells are normal in shape and size under the microscope. No leucocytosis. Red cells 2,760,000.

DESCRIPTION OF PLATE XVIII.

To illustrate Mr. Lunn's case of Facial Diplegia.

Adlard & Son, Imp.

XIV.—*A case of Facial Diplegia.* By JOHN R. LUNN. *Exhibited January* 12, 1900.

ANN C., æt. 51, was admitted on June 7, 1899, and is very deaf, thus adding to the difficulty of obtaining an exact history.

On examination it was found that her face was quite immobile, and no emotional movements of any kind could take place; skin smooth and devoid of wrinkles. She could close her eyes, but not quite completely. No inflammation of lids or cornea. Eyes rather watery; lower lids lax; both angles of her mouth lowered, giving the face a peculiar "sheepish" appearance. When drinking milk the glass is held firmly pressed against the lower lid, and the head moved as a whole. Cannot smile, frown, or whistle. Speaks loudly in a jerky way, slurring some of her words. Cannot show her upper teeth. Food apt to collect and stay in her mouth. Protrudes the tongue well and in the middle line. (December 26—cannot protrude tongue.) She can slightly move the ala nasi, but cannot apparently "sniff." No deviation of uvula, and no paralysis of soft palate. No facial herpes. Complained of pain in right side of face and head. There was no heart or lung mischief, and the urine contained a very faint haze of albumen, the quantity being normal.

The condition started suddenly (according to patient's account) on June 5, 1899, *i.e.* two days before admission. She has had a discharge from the right ear for some weeks before admission; no discharge from left ear. Both membrana are perforated. She cannot hear a sound at all, soft or loud. Has had vertigo for some time previously. At times complains of pain when the head is tapped, and hears "noises continually in head." Walks well, without reeling.

Nervous symptoms.—Knee-jerks normal, and other deep reflexes. No areas of anæsthesia. Taste, on admission, quite lost; cannot distinguish salt, sugar, or quinine on tongue. (This returned on July 20.) Smell unaffected. Eyesight normal.

No rise of temperature on admission.

Electrical reactions.—Faradism: muscles do not react at all except masseter muscle. Galvanism: at right L^{la}. of mouth A.C.C. greater than K.C.C. R.D. at $L^{la}.L^{la}$. A.C.C.

markedly greater than K.C.C. No response in frontalis muscles to galvanism. In the other facial muscles K.C.C. less than A.C.C.

The family history cannot be obtained. There is no history of rheumatism or syphilis.

Treatment.—Blisters and iodide of potassium and strychnia. Takes food well. Complains of vague pains in the extremities, and numbness of the fingers of the right hand.

From time to time patient has had rises of temperature, and has vomited. A purge has usually been found to reduce this.

Later remarks (December 26).—Cannot close eyelids properly; very blue sclerotics; when sound asleep the eyelids droop and very nearly close. Trismus very marked, due to (?) contraction of the muscles of mastication; no crepitus like that of osteo-arthritis on limited movement obtainable. There is no optic neuritis.

XV.—*A case of Malignant Goitre after two operations.* By Charters J. Symonds, M.S. *Exhibited January 12, 1900.*

Miss B., æt. 58, was first seen on September 30, 1897. She had a large, hard, nodular fixed growth in the left lobe of the thyroid; the right side was quite free. The thyroid cartilage was pushed to the right near the angle of the jaw. She made a loud noise on breathing, and the dyspnœa was increasing. The unilateral character suggested at the time that she was suffering from calcification of old cysts, the alternative diagnosis being that of carcinoma. The left vocal cord was fixed.

She gave a history of having a lump in the centre of the throat for nine years, without any difficulty in breathing. Seven years ago it reached the size of a walnut, and then was stationary until eighteen months ago, when it began to increase, and during the last six months had rapidly enlarged, with increasing dyspnœa. It was necesary to relieve her breathing, and the question lay between extirpation of the mass on the left and simple tracheotomy, or a combination of

the two. Having had some experience in operating on these malignant growths, I decided, in October, 1898, to remove the masses if calcareous, or if they proved to be growth to take away as much as possible. I was able to remove the greater part, and at the same time inserted a tube, which latter was removed on November 18, the breathing then being comfortable and free. The left cord was definitely paralysed as the result of this operation.

By January 13, 1899, a good deal of recurrence had taken place and the stridor had returned. In March, 1899, she spoke clearly and distinctly, but the cough was stridulous, and on deep breathing there was a little stridor. She talked quickly and freely, did her usual household duties, and had no trouble at night. The left cord remained paralysed; the right was normal.

On April 18, 1899, I again operated, removing large masses both from the upper and lower parts, without tracheotomy.

The case, I think, demonstrates the freedom with which these malignant growths may be handled, and the comparatively slight risk of hæmorrhage, for in neither of these operations was it of any moment at all; also the superiority of removal to tracheotomy. I would add that these remarks apply to the unilateral cases particularly.

At the present time the patient can breathe easily, though the stridor on exertion points, I think, to tracheal pressure. A mass was left behind the sternum at the second operation. She has a small fistula in the site of the tracheotomy tube, but no air escapes.

XVI.—*A case of Excision of the Neck of the Inferior Maxilla for Temporo-maxillary Ankylosis.* By J. Jackson Clarke. *Exhibited January* 12, 1900.

THE patient, a girl æt. 8 years, had scarlet fever with diphtheria three years and a half ago. She was treated at one of the Asylums Board hospitals in London. During the illness she had an abscess on the right side of the face, which has left a small scar near the lobule of the ear.

Three months after patient's return home the mother noticed that the jaw was stiff, and took the patient to one of

the London general hospitals. There an anæsthetic was given and an operation was done. This was probably an attempt to break down adhesions under chloroform. The condition was not relieved. The patient was next referred to the dentist to the same hospital, who removed the molar teeth from the left side of the mouth. A year later she was admitted as in-patient to the same hospital, but the surgeon decided to do nothing further, and the patient was discharged.

In November, 1899, the patient was sent to me at the North-West London Hospital. Her appearance at that time is shown in the photograph, in which the right side of the

FIG. 9.

face is seen to be much fuller than the left. The patient was admitted and prepared for operation. Under the anæsthetic, which was administered by Dr. Barford, anæsthetist to the North-West London Hospital, it was found that the ankylosis was absolutely unyielding, the molar teeth on the right side remaining in close apposition when an attempt to open the jaw was made. From the history of the case, and from a suspicion I had of some yielding on the left side during manipulation, I cut down over the right temporo-maxillary joint

through an incision planned to avoid the trunk of the facial nerve, the superficial temporal artery, the parotid duct, and other structures around the joint. About $\frac{5}{8}$ inch, *i. e.* the whole of the neck of the bone, was removed. The condyle itself, which was firmly synostosed to the temporal bone, was left alone. After excision of the bone on the right side the jaw was found to open and close easily, showing that over three years of complete fixation had not caused any important change in the joint on the left side. The patient experienced immediate relief, and on the third day was able to masticate food. When she left the hospital, fourteen days after the operation, she had gained 6 lbs. in weight, and at the same time she was satisfied with a smaller quantity of food than she had required when unable to masticate.

Dr. Wilfred J. Harris, who kindly examined the condition of the nerves for me six weeks after the operation, reports that "there is slight weakness in the right frontalis and orbicularis muscles. This is doubtless due to the dragging on the uppermost branches of the cervical division of the facial that was necessitated by a necessarily short incision, and I do not doubt it will pass away completely. The photograph shows the present range of mobility of the jaw. It will be noted that on extreme abduction the lower jaw deviates a little to the right. It is of advantage to this patient that operative treatment was adopted before the later consequences of the ankylosis; atrophy and recession of the lower jaw and displacement of the lower teeth to the horizontal direction had occurred.

XVII.—*A case of Friedreich's Paralysis.* By Arthur Voelcker, M.D. *Exhibited January* 12, 1900.

E. C., æt. 12 years, has been under my observation since August, 1896, when he was brought to the Hospital for Sick Children, Great Ormond Street, on account of his unsteady gait. Both parents are alive and well, and there is no family history of any nervous disorders. The patient is the third of four children; the other three are well.

When five years of age patient is said to have had German

measles and congestion of the lungs, after which he had shaking fits which were said by the medical man to be St. Vitus's dance. The fits of shaking are sometimes so marked that the patient is unable to feed himself. He falls about a great deal. He does not complain of headache, but the mother states that the child is liable to cramps in his hand after holding things, and then has "pins and needles."

When first seen in 1896 he was a rather undersized boy, who stood on a wide base and kept his head hung. There were irregular tremors of his head, tongue, and upper limbs, the left side being worse than the right. His gait was unsteady but showed no stamping, and he had well-marked Romberg's sign. There was no nystagmus. The pupils were equal and reacted to light and accommodation. No ocular palsy. No headache. Sensation to touch and pain normal and not delayed. Co-ordination not very good. A pin was picked up better with the right than the left hand. Speech was rather thick, but there was no elision. The knee-jerks were not obtained. There was no trouble with micturition. No muscular wasting. By the end of the year under treatment with iodide of potassium the tremor was less and the speech improved.

In April, 1897, his condition was practically unaltered, except that it was noted that he could see very little with his right eye.

In August, 1897, it was noted that he was unable to see with the right eye, and could only read letters 6 mm. long at a distance of two inches. His memory was excellent, his speech more distinct; he could touch the tip of his nose readily with the finger of either hand.

In November, 1897, there was a little ataxy of his arms, more marked on the left side. Ophthalmoscopical examination showed atrophy of the right discs, with old choroido-retinitis and some opacities in the vitreous body.

During 1898 he took cod-liver oil and iron for the first six months, and then iodide of iron, which he is still taking. His general health was good, and the unsteadiness unaltered.

In January of 1899 my colleague, Mr. Donald Gunn, kindly examined the boy for me, and reported that "the changes in the fundi are briefly those of an old syphilitic retino-choroiditis. The whole of the superficial choroid shows atrophic changes (nowhere affecting the deeper layers), and he has general sclerosis of the retina with (secondary) incomplete atrophy of the optic discs. All this points to his having been

one of the cases of retino-choroiditis at about nine or ten months old.

During 1899 his condition was unchanged, except that he had an attack of herpes labialis similar to attacks he had had in February, 1898, and August, 1897.

In November, 1899, it was noted that tickling the sole of the foot produced extension of the great toe.

For the last three weeks his unsteadiness is rather more marked, and his handwriting is more shaky.

XVIII.—*A case of Erythromelalgia.* By H. Batty Shaw, M.D. *Exhibited January* 12, 1900.

THE interesting feature of this case is the possible association of the erythromelalgia with an injury inflicted some years previously. The association is not a common one, but two cases have been described by Weir Mitchell and Lewin. For permission to show this case I am indebted to the courtesy of Dr. J. Rose Bradford.

Philip N., æt. 29 years, is at present engaged as a pointsman on a tram line. About ten years ago he was struck with a cricket ball on the back of the left wrist, and three weeks later an abscess developed in the site and soon healed; later on, however, two other abscesses developed, and after healing left a scar. For a few years the patient had no further trouble, but about three years ago he noticed the left hand and wrist becoming redder than natural, and a week or two later pain developed, and this pain became much aggravated by warmth. A short time after the development of the pain he noticed the hand swelled, and ever since he has complained of the pain, swelling, and redness, the latter sometimes giving place to a livid colour.

There is nothing in his history other than the above injury, and the patient can give no account of his parents. The pain is greatly relieved by cold, and he is much more comfortable in winter than summer. The pain also is much relieved by hanging the hand down; if the hand is raised he has much more pain. Voluntary movement of the limb also increases the pain. The limb as a rule is cold, but becomes warmer with exercise.

The hand as a rule looks reddish, but when very cold is purple, and latterly the patient has noticed that here and there mottled areas have appeared, some a brighter red, some blue. Occasionally the limb becomes lighter and darker in colour quite independent of exercise or applications; at these periods the limb swells more than usual. The hand is, under observation, becoming more swollen, and the dorsum pits readily; the skin has since last year become quite glossy, and the nails show transverse ridges such as are caused by trophic disturbance.

The pulse at the left wrist is not quite as easily felt as on the right side, but this probably is due to the swollen condition of the left wrist; there is no obvious arterial disease to be made out, though the patient complains of the tip of the nose becoming rather red and cold occasionally, and though there is some cyanosis of the face, ears, and feet.

Sensation is perfect without any hyperalgesia to heat and cold. The fingers and wrist cannot be used, only a slight movement being present. The reaction, however, to faradism is perfect, and there is no reaction of degeneration.

XIX.—*Non-recurrence five and a half years after Removal of the Tongue (Symphysis Menti) and Submaxillary Glands for extensive Epithelioma.* By Arthur E. Barker. *Exhibited January* 12, 1900.

THE patient, æt. about 45, presented himself at University College Hospital five and a half years ago for epithelioma, involving nearly the whole tongue and the bone of the chin. The glands under the jaw on either side were also obviously infected. The whole tongue and symphysis menti were removed by Mr. Barker by median incision through the lower lip to the hyoid bone. Some weeks later the glands on one side were removed, and later on those on the other.

The patient is now in excellent health, and has no evidence of recurrence anywhere. The jaw has united in the middle line; this has the inconvenience that the lower molar teeth are out of the line of the upper, and thus mastication is impossible. Otherwise the patient is comfortable.

DESCRIPTION OF PLATE XIX.

A skiagraph to illustrate Mr. Barker's case of Direct Fracture of the Patella.

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Microfilm

XX.—*A case of Direct Fracture of the Patella with no palpable displacement discovered by X rays.* By Arthur E. Barker. *Exhibited January* 12, 1900.

THIS patient, a man æt. 48, fell on the pavement striking his knee. As there was considerable pain he came to hospital. No fracture could be made out, but it was thought better to have a skiagram made. To the surprise of all who had examined the case a complete transverse fracture was thus demonstrated, with separation of the fragments of about $\frac{1}{3}$ inch. The ease with which such a fracture could be overlooked was in this way shown. Union took place without the use of a splint.

XXI.—*A case of Arthrectomy of Knee for Tuberculous disease, followed by restoration of function.* By Frederic Eve. *Exhibited January* 12, 1900.

ELIZABETH K., æt. 13, was admitted to the Evelina Hospital with a swelling of the right knee which was stated to have existed for about three weeks. The disease had almost certainly existed for a much longer period. The joint presented the ordinary appearances of tuberculous disease of the synovial membrane. The upper part of the synovial membrane could be felt distinctly thickened, but somewhat firm in texture. The lateral grooves on each side of the patella were filled up. Movement was limited and painful. On the anterior aspect of the limb, a few inches above and below the joint respectively, were two typical tubercular ulcers of the skin.

The patient was admitted on April 2, 1899. The limb was placed on a back splint, Scott's dressing with strapping was applied to the joint, and iodide of potassium with mercury was administered in order to exclude syphilitic disease. There was nothing indicative of congenital syphilis about the teeth, lips, eyes, skull, or nose, but her physiognomy was slightly suggestive of it; and on this ground only the

above mixture was given. No improvement taking place, on June 13, 1899, the operation of arthrectomy was performed. A transverse incision was made over the centre of the patella; a longitudinal incision was then carried down each lateral aspect of the limb, intersecting the ends of the transverse incision. The patella and capsule were then divided in the lines of the incision. The thickened synovial membrane was removed in the usual way, and that portion covering the posterior ligament of the joint was snipped away with scissors. The cartilage covering the most prominent part of the internal condyle was ulcerated. A slice was removed with a scalpel. The lateral and crucial ligaments were carefully preserved. The capsule was then united with thin silk sutures; the patella drilled, and the sawn surfaces drawn together with two thick silk sutures; and the skin wound completely closed with silkworm gut. Primary union took place.

July 25.—Under an anæsthetic I broke down the adhesions in the joint, flexing it to the full extent. The limb was then placed on a back splint, and an ice-bag applied. From that time, with some few exceptions, the limb was removed from the splint every day, and massage and passive movements were carried out. Owing to a return of stiffness the adhesions were again broken down under an anæsthetic on August 10, 13, 26, September 6, 22, and October 4. The movements became gradually freer, and when she was discharged to a convalescent home on November 9 she could walk with a scarcely perceptible limp. When shown at the Society she walked without a limp. The knee could be fully extended, and could be flexed to a right angle. There was no displacement backwards of the tibia, and the joint presented a normal appearance.

Less trouble would probably have arisen in obtaining restoration of function if the passive movement had been commenced somewhat earlier, for example a month after the operation.

The joint disease was of a subacute character, and although there was ulceration of the cartilage there was no extensive caseous degeneration or softening of the synovial membrane. Nevertheless the disease was distinctly progressing, although the usual treatment by rest and pressure was given a fair trial. The operation was undertaken with a distinct view of obtaining restoration of movement, and to this end the details of treatment were adapted.

DESCRIPTION OF PLATE XX.

Illustrating the Range of Movement in Mr. Eve's case of Arthrectomy of the Knee-joint.

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XXII.—*A case of Myeloid Sarcoma of lower end of radius; resection of lower ends of radius and ulna with good functional result.* By FREDERIC EVE. *Exhibited January* 12, 1900.

MRS. T., æt. 21, a young married woman, fell and sprained her right wrist in June, 1898. The joint remained weak and painful, and in September she noticed a swelling above the joint which gradually increased.

February 27, 1899.—I saw the patient in consultation with Mr. Hughes Davies. The lower fourth of the right radius was uniformly enlarged by a central tumour, the surface of which was smooth, in parts elastic, and egg-shell crackling could be obtained. The circumference was an inch and a half greater than that of the left wrist. The movements of the joint were free, and the synovial membrane unaffected.

The radiograph (see Plate XXI, fig. 1) clearly shows that the tumour is central.

A diagnosis of myeloid sarcoma was made, and resection recommended.

March 4.—Assisted by Mr. Hughes Davies, I exposed the lower ends of the radius and ulna by an incision on each side, and removed an equal length of each bone. The wrist-joint was unaffected. Each individual tendon was drawn out, a portion cut from it corresponding roughly to the length of bone excised, and the ends were united with silk.

No large vessel was injured. The wound was closed and the limb placed on an angular splint. Primary union took place, the temperature never rising above 99°.

Six weeks after the operation the patient was able to write her name. She is now able to write a firm even hand, to sew, to cut her food at meals, and to lift any light article, such as a glass. There is also good power of flexion and extension of the wrist. The flexion of the fingers is, however, somewhat limited, but this I trust will improve. She was seen on February 21st, 1900. An improvement in the firmness and mobility of the wrist had taken place. The accompanying photographs (Plate XXII) were taken at that time.

The tumour is a typical myeloid sarcoma, and is surrounded

with a thin but complete capsule of bone. The articular cartilage was as usual intact. The substance of the tumour had largely undergone cystic degeneration, and had not to any appreciable degree extended up the medullary cavity beyond the point where the enlargement of the shaft commenced.

A section under the microscope showed large numbers of multi-nucleated giant-cells lying in a tissue composed of round cells with a homogeneous or fibrillar matrix.

Mr. Annandale has recorded* a case of myeloid sarcoma of the radius treated by resection of the affected portion. He kindly writes me as follows:—"The patient was seen eight years after the operation, and the hand was most useful; an occupation as an engineer was carried on quite successfully."

Mr. Clutton reports a similar case,† and in his paper may be found references to three other cases in this Society's *Transactions*.

I am not aware that shortening of the tendons has in previous cases been combined with resection of the radius and ulna.

XXIII.—*A case of Primary Tuberculosis of the Nasal Septum (first shown at the Society in October*, 1897). By StClair Thomson, M.D. *Exhibited February* 23, 1900.

THIS patient was shown to the Society two and a half years ago, and her history may be epitomised as follows. Four and a half years ago she commenced to suffer from nasal discharge and blocking of the right nasal passage. She consulted Dr. Watson Williams of Bristol in July, 1896, who found a smooth, bluish-pink, sessile growth the size of a filbert on the right nasal septum. There was some crusting of the mucus in the left nasal passage, but there was no obvious disease. The appearance of the growth raised the suspicion of sarcoma, but when removed and examined histologically it was found to be a tuberculous growth, though no

* *Brit. Med. Journal*, Dec. 10, 1881.
† *Trans. Clin. Soc.*, vol. xxvii, p. 93.

DESCRIPTION OF PLATES XXI AND XXII.

Illustrating Mr. Eve's case of Myeloid Sarcoma of the Radius.

PLATE XXI, fig. 1, is a skiagraph before operation. Fig. 2, after operation.

PLATE XXII exhibits the range of movement.

FIG. 1.

FIG. 2.

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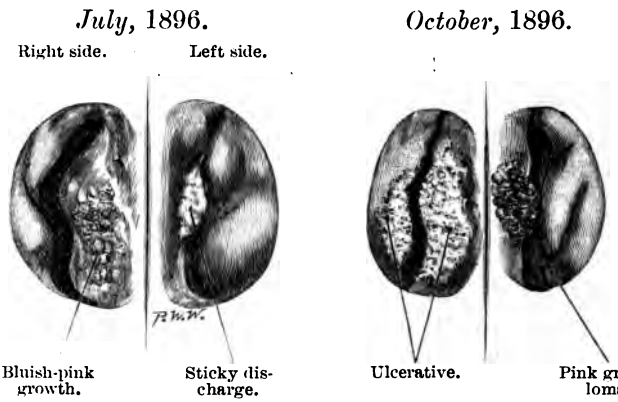
bacilli were present. When seen three months later the tuberculous deposit had spread through to the left side, appearing there as a pale pink uneven growth (Fig. 10). At the Bristol Royal Infirmary she was treated with repeated curetting, and applications of lactic acid to the septum and inferior turbinal on either side. She also had three injections of Koch's tuberculin about three and a half years ago. Six months later a tuberculous nodule was noticed on the posterior pharyngeal wall.

This latter was evident when she came under my observation three years ago, and it was still present, although reduced to the size of a threepenny piece, when I showed her to the Society in October, 1897. At that time the perforation

FIG. 10.



in the septum was irregularly circular, and large enough to admit a cedar pencil. This opening in the triangular cartilage was surrounded by pale, ulcerating nodules. One of these was removed, and the sections—which are placed under the microscope—show it to be infiltrated with tubercle, the giant-cell systems being well marked with foci of caseation. Sections were stained and examined for tubercle bacilli, but after prolonged search no bacilli were discovered. In addition to the perforation of the septum and the breaking down round its margin, there was pale indolent ulceration of both inferior turbinals.

The treatment has been limited to cleansing the nose with simple alkaline lotions, and the application of Mandl's solution of iodine and iodide of potassium to the pharynx and

the nasal mucosa. Later on a 50 per cent. oily solution of guaiacol was tried, and appeared to be beneficial.

Soon after she was examined here the patient returned to the country, and only came under my observation a short time ago. It will be seen that she presents no very obvious symptoms. Her weight is 8 st. 8 lbs., instead of 9 st. as on the former occasion, but she has neither cough nor fever, and I find no trace of mischief in her lungs or larynx. On examining the interior of her nose it is seen at once that the perforation has decidedly increased in size. It is now irregularly ovoid, extending an inch horizontally and about half an inch across. It is still limited to the cartilaginous septum. Its margin is thick, pale, tubercular, and the neighbouring mucous membrane is infiltrated and is breaking down. But while this spread of the disease has taken place it is remarkable to note the improvement elsewhere. There is now no infiltration or ulceration visible on the inferior turbinals, while the ulceration on the posterior wall of the pharynx has completely healed, leaving only a dry and somewhat atrophic surface.

When the patient was previously shown to the Society there was some doubt raised as to its being of a tubercular character, since the tubercle bacilli had not been discovered. At that time I pointed out that the patient had reacted strongly to tuberculin, and that this was generally accepted as indicative of tuberculosis. I might now add that in the cases recorded by other observers the bacillus has only been found in some instances.

By a process of exclusion I cannot see that this slow-spreading affection limited to mucous membrane and cartilage can, in the nose, correspond to any other condition.

If it is agreed that the disease is tubercular, then we have here one of the rarest manifestations of tubercle. There is, I believe, no part of the whole respiratory tract which is so seldom attacked as the Schneiderian membrane, and this fact is a clinical confirmation of the strong defensive arrangements of the nose which I have helped to elucidate. I have been informed that no other case beyond the present one of primary nasal tuberculosis has been put on record by a British observer. Even in cases of pulmonary tuberculosis it is very rare, I believe, for the nasal mucosa to be affected, although that of the larynx is so frequently involved. The post-mortem statistics of Willink* show that in 1600 autopsies he only met

* Prager, *Vierteljahrschrift f. prakt. Heilk.*. Bd. xxxviii, 1853.

with one case where the septum was affected, although there was pulmonary tuberculosis in 450 instances (28·1 per cent.).

No surgical measures have been carried out in this case, and it is interesting to note that the hope I expressed two and a half years ago of spontaneous healing has been realised in part. The thinness of the mucous membrane over the septum and the deficient vascular supply possibly account for the progress of the disease in this region.

I should like to have the opinion of members not only as to the diagnosis, but also as to what they would suggest for treatment. Measures which are successful in other forms of accessible and localised tuberculosis might be useful here. If the treatment by tuberculin is really of service this is surely a suitable case.

XXIV.—*A case of Scoliosis in an Infant.* By J. Jackson Clarke. *Exhibited February* 23, 1900.

THE patient, Emily V., is one year and five months of age. She was first brought to me when twelve months old. Then as now she had a well-marked deformity convex to the right in the dorsal, to the left in the lumbar region. Combined with the scoliotic curve is a considerable amount of dorsal kyphosis. The mother first noticed the deformity when the patient was thirteen months old.

When I first saw the child she had pronounced rickets, but was fairly strong muscularly in spite of the epiphysial swellings being pronounced. The mother has been in the habit of nursing the child on the right arm. Infantile scoliosis, whether due to rickets or congenital, is not uncommon. If it is left untreated many of the patients become hopelessly deformed and disabled, and when they are grown up have a pulse rate of about 130, and respiratory rhythm of 36. In addition to diet and medical treatment, daily manipulations, and attention to the mode of nursing, instrumental treatment is in my opinion necessary in these cases. The form of instrument that I have found of most service is the simpler form of Chance's splint. Although a rigid splint it only exerts pressure on the unduly prominent parts of the spine, and thus not only acts like an effective straight splint

in rickety deformities of the limbs, but also affords the only form of so-called "muscular education" that an infant is susceptible of. Prolonged recumbency, and apparatus such as the poroplastic jacket, I have found to cause a loss of general health and strength, and muscular enfeeblement.

XXV.—*A case after Open Section of the Knee-joint for Irreducible Traumatic Dislocation.* By W. H. Battle. *Exhibited February* 23, 1900.

THE patient, a married woman æt. 50, was admitted to St. Thomas's Hospital on July 23, 1898, and left on October 8 of that year.

She was admitted for an injury to the right knee sustained during an attempt to enter a train in motion. She fell between the train and platform, and her leg was violently twisted outwards. There was a curious appearance of the limb, which looked in a condition of slight genu valgum. The skin over the inner aspect of the joint was puckered and bruised, and there appeared to be an unusual separation between the inner condyle of the femur and inner head of tibia. The patella was displaced outwards, and although not fixed could not be restored to its normal position. The knee was kept partially flexed, and could not be voluntarily flexed or extended by the patient. The leg could be freely abducted, showing complete rupture of the internal ligament; whilst the free movement from before backwards on pressure over the tibia when the knee was flexed under anæsthetic showed that the crucial ligaments were ruptured.

Attempts had been made to reduce the displacement of the bones before I was asked to see the case, without success. Examination with the X rays showed some separation between tibia and femur on the inner side, and a partial displacement of the patella outwards. No improvement having followed manipulation, it was decided to open the joint and do what might be possible to repair the injuries sustained.

On August 5 a free incision was made across the front of the joint dividing the ligamentum patellæ, and the interior of the joint exposed. It was found that the difficulty in replace-

ment of the patella was caused by the interposition of a thick muscular and fibrous band, which had been torn from the inner condyle and become stretched between it and the patella; the condyle was caught almost like a button by the button-hole, and the increased distance of the femur from the tibia was due to interposition of part of this torn tissue. As it could not be readily reduced, it was cut across and then cleared from under the patella, so that that bone could return to its normal position. The joint was more fully opened and the crucial ligaments came into view: the anterior one had been torn away from its attachment to the femur, leaving sufficient ligamentous structure attached to admit of sutures being inserted; the posterior was not completely torn off. Silk sutures were passed to restore these ligaments, and afterwards the internal lateral ligaments and the ligamentum patellæ were sutured also with silk, and the wound closed without drainage.

There had been little hæmorrhage into the joint, and there was little effusion, the knee being almost dry. The puckering of the skin resulted from the dragging of the structures which had been pulled outwards under the patella.

Recovery was for a time retarded by suppuration in the inner side of the wound, but it remained localised.

The knee is apparently normal as regards its ligaments, can be flexed to a right angle, and the patient walks well.

XXVI.—*A case of Achondroplasia.* By Thomas H. Kellock. *Exhibited February* 23, 1900.

THE patient, a boy aged 4½ years, is a typical example of the congenital condition termed achondroplasia, or chondrodystrophia fœtalis hyperplastica; two cases of the same condition have been shown at this Society, one by Dr. Garrod in 1898, the other by Mr. W. Turner last year, and to both those cases the patient now shown bears a very strong resemblance.

The father and mother of this child are working people living at Bethnal Green. They were no relation to each other before marriage. The mother has one sister, the father four brothers and three sisters; they are all normally developed, as are also their children. There is no family history of de-

formity or maldevelopment to be obtained, nor can the mother account in any way for the child's condition.

There are four children in the family :

The first—male æt. 6 years, born eleven months after the parents' marriage, normal.

Second—the patient shown, æt. 4½ years.

Third—female æt. 2½ years, normal.

Fourth—male æt. 6 months, normal.

The patient was born at full term ; the mother had been well and had not suffered any privation during the pregnancy. The labour was easy, but directly the child was born it was noticed that he was a queer shape and very fat ; he was breast-fed only until ten months old. The mother states that it was not until he was six months old that he began to crawl

FIG. 11.

about the bed ; this she considered very late : he did not walk until he was two years old. He now shows markedly the typical features of the condition : he is only 30½ inches in height, as compared with 39½ inches, the height of a normal child of the same age, and almost the whole of this deficiency is to be found in the legs ; the spine, measured from the vertebra prominens to the tip of the coccyx, being almost of normal length. The long bones of the limbs are very much shorter than normal ; the epiphyses at the ends of these bones are abnormally large. The shape of the head with its large vault and narrow base, the lordosis, prominent abdomen, genu retrorsum, waddling gait, and peculiar manner in which he raises himself from the sitting position and assumes it again, the peculiar divergent position of the fingers, are all features which have been pointed out in the previous cases. Like them, too, the child is perfectly intelligent ; his skin, hair, and muscular development, as well as his general health, are quite normal.

DESCRIPTION OF PLATE XXIII.

To illustrate Mr. Kellock's case of Achondroplasia.

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In a paper published in the *Practitioner*, 1899, Mr. William Turner gives a full description of his case, with some excellent photographs and skiagrams, and he mentions two features in his case which are noticeable in this child,—that the arms cannot be fully extended at the elbows, and that there is considerable laxity of the ligaments of several of the joints, allowing abnormal lateral movements; this is especially noticeable in the knees.

The following table gives a few measurements compared with those in a normal child of the same age:

	H. J., achondroplasia. Inches.	Normal child of same age. Inches.
Total height	30½	39½
From vertebra prominens to tip of coccyx	11½	13
From acromion to tip of middle finger	11	17
From acromion to external condyle of humerus	4½	6½
From anterior superior spine of ilium to internal malleolus	11½	19
From anterior superior spine of ilium to bottom of internal condyle of femur	5¾	10¾
Circumference of head	21	20
Girth at umbilicus	22	18½

XXVII.—*Two cases (father and son) of Deformity of the Hands with Absence of a Radius in the child.* By Charles W. Chapman, M.D. *Exhibited February* 23, 1900.

THE father, who is an assistant at a tailor's shop, presents the following conditions:

Right hand.—There is no deficiency of bones and no club-hand. The metacarpal bones of the thumb are well developed; the terminal part has three phalanges, the second being flexed at a right angle on the first, and the third on the second. The thenar eminence is almost entirely absent, and adduction and abduction are probably non-existent.

Left hand.—The terminal portion, that which represents the thumb, is united for two thirds of its extent to the index finger; it has three phalanges as on the right hand, and only a slight degree of contraction of the joint between the first and second phalanges. There is a wide cleft between the

first and second fingers, and the man appears to use the index finger as a thumb rather than a finger. The power of adduction in the index finger appears to be due to a strong development of the dorsal interosseous muscle.

The patient can write and shave,—in fact, he can do most with his hands like other people.

The son is an intelligent child of three years of age.

Right arm.—The bones of the forearm appear to be perfect. The thumb has a small but complete metacarpal bone; at its terminal part it resembles a finger; it has three phalanges, the second and third being permanently flexed on the first. There is power of adduction and abduction of the thumb, and the thenar eminence is fairly well developed.

Left arm.—The congenital defects are striking. There is entire absence of the radius, and there is a club-hand of the radio-palmar variety, *i. e.* the hand is in a position of pronation and complete abduction. The lower end of the ulna appears to be of normal size, but it is very prominent at the wrist. The carpal bones are displaced outwards. The first metacarpal bone of the thumb is entirely wanting, and there is congenital contraction of the middle finger.

The child can feed himself and can hold a pencil, but he is unable to button or unbutton his clothes.

There has not been a similar case shown before this Society, though four cases of deficiency of the tibiæ have been exhibited. One by Dr. W. H. White is reported in vol. xxi of the *Transactions* four and a half years of age, and three cases exhibited by Mr. Clutton and recorded in vol. xxix of the *Transactions*. In none of these was there any deformity in the other member of the family.

Mr. Hutchinson has a case of congenital absence of both radii reported in the *Medical Press and Circular* for July, 1895. The family history is interesting. The mother had twice married; she had had five children by the first husband, of whom two were deformed and died in infancy. By the second husband she had had four children, all of whom were deformed. Mr. Tubby, in his work on deformities published in 1896, summarises the records of cases of congenital absence of the radius, in which it appears that forty-five cases had been recorded, in twenty-six of which the deformity was bilateral. The children were for the most part premature and still-born, and the remainder seldom lived many weeks.

In the present instance the child is quite strong and healthy, also intelligent.

DESCRIPTION OF PLATE XXIV.

A skiagraph to illustrate the Deformity of the Hands in Dr. Chapman's Case (that of the Father).

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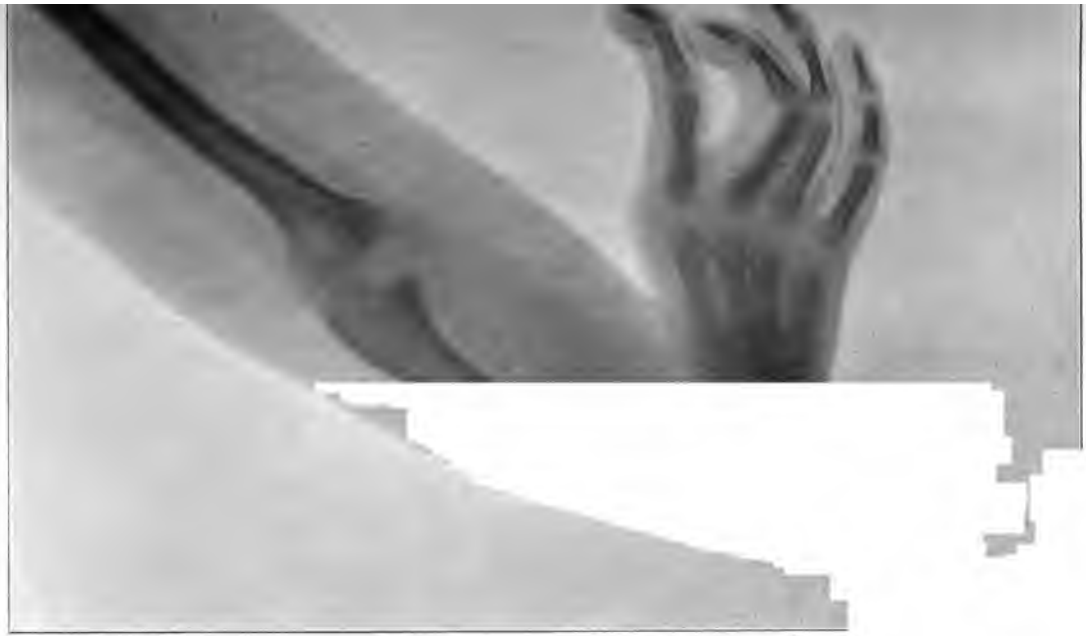
DESCRIPTION OF PLATE XXV.

A skiagraph showing Absence of the Radius and Deformity of the Hand in Dr. Chapman's Case (that of the Son).



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XXVIII.—*A case of Asthenic Bulbar Paralysis (Strümpell's).* By LEONARD GUTHRIE, M.D. *Exhibited February* 23, 1900.

FEMALE æt. 23, waitress, used to work twelve hours daily. Two years ago difficulty in articulation, occurring when fatigued. Eight months ago noticed difficulty in deglutition and mastication, and inability to fully close eyes. No history of sore throat.

Present symptoms.—Weakness of orbicularis oculi and orbicularis oris, of the tongue, pharynx, and soft palate. Cannot fully close eyes or resist passive elevation of eyelids. Cannot whistle or blow up cheeks unless anterior nares are closed and her mouth supported. Cannot empty spoon with her lips. Food collects in right side of cheek, and she has lost control over movements of tongue. Cannot curl tongue transversely, thus showing association between orbicularis oris and transverse lingualis muscles.

Movements of soft palate are defective, and of the pharynx. She chokes over solids, and liquids regurgitate through nose. The voice is nasal and indistinct in sounding explosive labials—B, P, and gutturals.

All symptoms vary greatly in severity, and are most marked when she is fatigued. She swallows breakfast well, but has difficulty with other meals. After reading a few lines aloud speech becomes almost unintelligible.

Reflexes, electrical reactions, and sensation are normal. There are no tremors, no wasting, and she has no pain.

She has occasionally weakness of fingers of right hand, preventing her from sewing.

Remarks.—The case appears to belong to the class called by Strümpell "asthenic bulbar paralysis." A somewhat similar case was shown before the Society by Dr. Harry Campbell in February, 1899. The remission in the symptoms, normal nature of reflexes, and electrical reactions are in keeping with the absence of pathological changes hitherto discovered.

XXIX.—*A case of Paralysis of the Seventh, Eighth, Ninth, Eleventh, and Twelfth Cranial Nerves of the left side.* By C. E. Beevor, M.D. *Exhibited February* 23, 1900.

SIX years ago contracted primary specific sore, followed by sore throat and rash. Two and a half years ago left facial paralysis and polypoid granulations in the aural meatus. Three months ago neuralgic pains in the left temple; in the left ear began six weeks ago. Voice became hoarse, and difficulty of swallowing arose. At present:

Seventh.—Left facial paralysis, including platysma.

Eighth.—Left ear: deafness to all forms; polypus in the meatus. Left side of tongue: taste lost front and back.

Ninth.—Some anæsthesia of left half of pharynx. Difficulty in swallowing.

Tenth.—No alteration of pulse.

Eleventh.—Paralysis of soft palate to volition and reflex action, and of left vocal cord, which lies in extreme rigidity. Paralysis of left sterno-mastoid and left trapezius, except a few fibres arising from sixth cervical spine to second dorsal.

Twelfth.—Tongue atrophied on the left, with fibrillar tremor, and goes to left on protrusion.

The depressions of the hyoid are not affected. The affected muscles do not react to faradism. Right optic neuritis, partial.

Probably syphilitic meningitis affecting nerves of medulla.

XXX.—*A case of a Lesion of the left side of the Pons and Medulla (? an initial lesion of Disseminate Sclerosis).* By James Collier, M.D., for Hughlings Jackson, M.D. *Exhibited February* 23, 1900.

A MAN æt. 40 years, who has never had a day's illness, and has not had venereal disease. Onset of illness gradual.

Symptoms in the order of their development: giddiness, deafness of left ear, alteration of articulation, difficulty in guiding the left arm and leg, diplopia, difficulty with swallowing and mastication, loss of feeling in left side of face; no headache, vomiting, nor dysuresis.

Summary of case.—Optic discs pale; paralysis of the fifth, sixth, seventh, and eighth cranial nerves on the left side; weakness of the ninth, tenth, and eleventh cranial nerves on the left side. Head held in right cerebellar position. Extreme left-sided hemiataxy. Slight spasticity of the right arm and leg.

Articulation resembles that frequently found in cases of disseminate sclerosis.

XXXI.—*A case of Macroglossia due to Lymphangioma.* By Arthur E. Barker. *Exhibited February* 23, 1900.

THE patient, æt. 16, came to me thirteen years ago with a typical lymphangioma of the tongue, producing great enlargement and inconvenience. It had been rapidly increasing of late, and began to chafe against the lower incisor teeth. Two lateral flaps meeting in the middle of the tongue far back were made, and the anterior half of the tongue was removed. The two lateral flaps of fairly healthy tissue were then sutured together along the middle line. Union took place to my entire satisfaction, and an almost normal-pointed organ was the result. I have frequently seen the child at intervals of a year or so since, and up to recently only a certain sponginess about the new frænum betrayed the presence of lymphangiomatous tissue. But lately this has begun to increase, and at the beginning of this year the girl came to me again asking for further operation, as the enormous tongue was most inconvenient. It was now thickened vertically, and had the usual appearance. This time I removed the anterior two thirds by two horizontal cuts, meeting far back in the middle of the organ. The upper flap was then stitched to the floor of the mouth at either side and in front. The result is satisfactory, except that the tip of the organ is some-

what fixed. Perhaps some plastic operation may remedy this condition.

The original growth I described in the *Transactions of the Pathological Society* ten years ago (vol. xli, 1890, p. 77). It was a typical lymphangioma, with some admixture of ordinary nævoid tissue. The present growth shows that the nævoid tissue has now got the upper hand to some extent, and there is more blood in the enlarged spaces than in the first specimen.

XXXII.—*A case of Aortic Regurgitant Murmur heard in the Pulmonary area.* By Norman Dalton, M.D. *Exhibited April* 27, 1900.

A MAN æt. 26. He was quite well until he had scarlet fever twelve years ago, with nephritis and some heart affection. Since then he has not been able to work hard on account of his heart. No rheumatic fever. Three years ago he was in King's College Hospital under Dr. Duffin, and the cardiac physical signs and the renal condition were then exactly what they are now.

The patient, although twenty-six, is quite hairless except on the head, and looks undeveloped. He is very pale. There is marked pulsation of the carotids; and the radial pulse, though small, is collapsing, as is found in aortic regurgitation complicated by mitral regurgitation. The apex-beat is in the fifth left interspace, one and a half inches external to the nipple line in the erect posture. The area of cardiac dulness is increased, extending externally from the third left costo-sternal articulation in a curved line to the apex-beat; while the inner border runs down the left border of the sternum as it should. There is comparative dulness in the second left interspace close to the sternum. The cardiac dulness varies slightly with the respiratory movements. There is a prolonged systolic murmur at the apex. At the third left costo-sternal articulation and in its vicinity a systolic murmur, a diastolic murmur, and an accentuated second sound can he heard. The systolic bruit can be heard to the right of the sternum, but the diastolic bruit can be scarcely heard there, and it is

not conducted downwards. On the other hand, both systolic and diastolic murmurs are conducted upwards and to the left, being loudly heard under the clavicle. They are also conducted to the back, being audible all over the back of the thorax; perhaps louder to the right of the vertebræ than to the left. There is a diastolic thrill in the second left intercostal space. Above the left scapula the second cardiac sound is accentuated.

The presence of the diastolic murmur in the situation (third left costo-sternal articulation) at which sounds made at the pulmonary valve are usually heard would suggest that there was pulmonary regurgitation, and that the case was one of congenital heart disease. This view might be supported by the fact that the development of the patient is imperfect. But, on the other hand, there is good evidence to show that the diastolic bruit, although in a wrong position, is due to aortic regurgitation. First we know that the case is not congenital,

because the patient was quite well until he had scarlet fever at fourteen years of age. Then there is no cyanosis. Then, in favour of aortic regurgitation, we have the pallor, the pulsation of the carotids, and the fact that the pulse, although small, is collapsing. The hypertrophied left ventricle would also indicate aortic disease; but this piece of evidence is discounted by the fact that the patient has a contracted kidney, the kidney apparently never having recovered from the scarlatinal nephritis. Lastly, the enlargement of the spleen, which is very obvious and is almost certainly due to infarctions, is in favour of a lesion of the aortic valves. I can only explain the abnormal position of this diastolic murmur and its abnormal lines of conduction by supposing that the heart became displaced when the patient was first ill, and then became fixed at the left side of the sternum by adhesions. These may have been pleuritic, but I think it quite possible that there may have been pericarditis, due to the scarlet fever itself or to the nephritis, and that the resulting adhesions and

extra-pericardial cellulitis may have fixed the heart in this very unusual position. However, it is clear that if the heart, as a whole, be situated to the left of the sternum, the aortic regurgitant murmur would be heard at the third left costo-sternal articulation instead of at the right. But it is more difficult to understand the alteration of the line of conduction of the bruit, viz. that it is not conducted downwards but upwards and to the back. It is true that, with the heart entirely on the left side, the ascending aorta would be stretched and more horizontal than normal, and consequently its axis would not be practically in a line with the axis of the left ventricle, but at an angle with it. This would certainly alter the line of conduction of a regurgitant bruit, and very likely permit it being directed downwards; but this does not explain why the diastolic murmur should be so loud in the back and even under the clavicle.

The presence of the accentuated second sound at the third left costo-sternal articulation is curious, and rather obscures the diastolic bruit. It might be pulmonary, due to the mitral regurgitation, but there is practically no back pressure of importance. There is no dropsy; and the urine, although albuminous, is pale and in good quantity, showing that no mechanical congestion is present to mask the characteristic urine of a contracted large white kidney. The lungs, also, suffer very little. Sometimes there is a little crepitation at the bases, and once or twice he has had an infarction in them with some fever; but there has been nothing of consequence. So it is quite possible that, in spite of the regurgitation, the accentuated second sound may be made at the aortic valves by the projection into the orifice of a rigid pocket, which is not met by its fellows and therefore fails to prevent regurgitation, but resists the shock of the returning blood, so as to make a loud noise. This view is favoured by the presence of a diastolic thrill in the same position as the sound; and so long as any part of the semilunar valves obstructed the return of the blood into the left ventricle the renal disease would tend to make the second sound accentuated.

The case is further interesting as being one of endocarditis undoubtedly due to scarlet fever, or perhaps to scarlatinal nephritis.

DESCRIPTION OF PLATES XXVI AND XXVII.

PLATE XXVI.

A skiagraph of Dr. Habershon's case of Tubercular Glands in the Anterior Mediastinum. The shadows to the left of the manubrium appear to indicate glandular enlargement.

PLATE XXVII.

A skiagraph from a healthy boy of about the same age, to contrast with the preceding plate.

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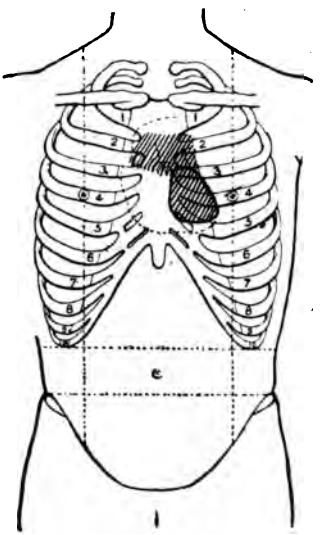
XXXIII.—*A case of Probable Tubercular Glands in the Anterior Mediastinum.* By Dr. HABERSHON. *Exhibited April* 27, 1900.

R. P., a boy æt. 8, had inflammation of lungs three years ago following measles. No other previous illness. No family history of tubercular disease. Was taken ill suddenly on August 1, 1899, with glandular swellings in both cervical regions and great febrile disturbance. So acute was the onset that it was at first mistaken for mumps. The glands

FIG. 12.



speedily suppurated, and on November 1 and November 10 Mr. S. Paget at the West London Hospital operated. The linear scars of these operations are seen on both sides of the neck.

Recently the boy has not been well. Several small glands have appeared in the neighbourhood of the scars, as well as in the lower cervical region. He has lost flesh slightly, complains a little of dyspnœa on exertion, and generally seems weakly. The temperature at night varies between normal and 99°.

The question of interest is whether the physical signs warrant the diagnosis of an extension of tubercular glands to the mediastinum, and if so whether the recurrent glands in the neck should be left alone.

Physical signs.—There is slight impairment of resonance for about a finger's breadth to the left margin of the sternum in the first and second intercostal spaces (left). This impairment seems to be continued across the manubrium sterni. Auscultatory percussion shows that the edge of the upper lobe of the left lung does not extend quite to the sternal edge. The lungs are healthy. The boy presents the signs described by Dr. Eustace Smith of a venous bruit over the area of impaired resonance when the head is thrown back. The above area becomes resonant when the boy lies down.

Radiographs have been taken, but are not decisive.

XXXIV.—*A case more than Two Years after the Removal of a recurring Tumour of the Frontal Region of Carcinomatous Character, and of the underlying portion of the skull.** By William Henry Battle. *Exhibited April* 27, 1900.

THE patient, a married woman æt. 37, had been under treatment at the Royal Free Hospital in January, 1898, for a tumour of the forehead. It was a recurrent growth, the first manifestation having been met with eight years previously. After various treatment the tumour was the size of a pea two years ago, when it again commenced to grow.

The tumour was of large size, measuring three inches from above downwards and four inches and a half from side to side, and projected as much as three and a half inches from level of the skin. It was an irregular fungating growth uncovered by skin, was a great disfigurement, and bled rather freely at times. There was no pulsation or enlargement of glands. The patient was thin, anxious-looking, and anæmic.

Operative measures were carried out in three stages:

1. An elastic bandage was put round the head to control the hæmorrhage; the soft part of growth was then removed

* See *Clin. Soc. Trans.*, vol. xxxii, p. 127.

DESCRIPTION OF PLATE XXVIII.

To illustrate Mr. Battle's case of Tumour of the Frontal Region.

One photograph shows the tumour before removal, the other the condition of the patient when exhibited.

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from the bone and the vessels secured. As the bone had been perforated it was removed later at—

2. A second operation by means of trephine and circular saw worked by a hand motor, aided by a few touches with a chisel.

3. Thiersch's grafts were used to cover in the exposed dura mater after granulations had sprung up.

The growth is a spheroidal-celled carcinoma, and the specimen is in the museum of the Royal College of Surgeons.

The deformity resulting from the removal of the growth is hidden by the patient, who combs her hair across the forehead. She is in good health, and has a healthy infant of six weeks old.

XXXV.—*A case of Recklinghausen's Disease.* By H. Campbell, M.D. *Exhibited April* 27, 1900.

A GIRL æt. 15. Two years ago had to take to bed in consequence of paraplegia from Pott's disease. Patient is very anæmic. The glands in the axillæ and supra-clavicular regions are enlarged. In certain regions, but especially in the right humeral region, the right posterior thorax, and to a less extent the left humeral region, are to be felt nodular masses, apparently consisting of hypertrophied subcutaneous tissue. They are somewhat tender. The skin in these regions is very loose, being most so where the nodular masses are most pronounced. The skin over the right forearm, however, is very loose, and here there is little subcutaneous hypertrophy. The skin is curiously pigmented. The pigment is deposited in patches varying in colour from light yellow to dark brown, and in size from a pin-point to several square inches. It is most marked over the subcutaneous masses. Thus the entire right humeral region is coloured brown, the colour being deepest where the masses are thickest. The trunk is also pigmented, chiefly in the form of minute spots, which show a decided tendency to symmetrical arrangement. Thus on the abdomen there are two spots about the size of a florin, one on either side of the middle line above the umbilicus. There is practically no pigmentation in the lower extremities.

When the patient was first admitted to the home, now six

years ago, the condition described was limited to the right humeral region, which presented much the same appearance as the left humeral region does now, *i. e.* there was a moderate degree of hypertrophy over the triceps, the overlying skin being pigmented; it was not till about a year ago that this latter region became involved. Since admission the disease has steadily advanced. The right hand is congenitally deformed. The third and fourth fingers and their metacarpal bones are fairly normal, but all the remaining phalanges and metacarpal bones are remarkably mobile, as though their several joints were devoid of ligaments, and were not fixed by tendons and ligaments. The hand has a flattened and splayed appearance. The first and second fingers are spindle-shaped. The patient is perfectly intelligent. She reads and does fancy work, though with difficulty, owing to the condition of the right hand.

XXXVI.—*A case of Successful Excision of a Subclavian Aneurysm.* By H. A. Lediard. *Exhibited April* 27, 1900.

THOMAS N., æt. 39, a fireman from Whitehaven, was sent to me by Dr. Harris on April 18, 1899, on account of an aneurysm of the subclavian artery.

History.—Patient's attention was first attracted by a throbbing sensation and slight swelling on the right side of the neck about ten months ago, but this caused him no inconvenience at first, and he did not attach much importance to it. He found, however, that the swelling increased steadily, and that his right arm was becoming gradually weaker, while he had occasional twinges of pain shooting down to the finger tips. These symptoms grew gradually worse, and at last he was obliged to give up work altogether. He then consulted Dr. Harris.

On admission to the Cumberland Infirmary there was a distinct swelling visible in the right supra-scapular space, about the size and shape of a duck's egg, pulsative and expansive, and evidently aneurysmal in character. The tumour rose above the centre of the collar-bone, and pulsation was felt below the collar-bone at the outer third.

There was a distinct systolic thrill over the aneurysm, but the cardiac sounds were normal. The external jugular vein was distended.

The right radial pulse was slightly weakened and delayed, but there was no œdema or wasting of the arm.

First operation—ligature of first part of subclavian.—On April 14 the first part of the right subclavian artery was tied with a broad flat ligature of kangaroo tendon.

The vessel was exposed close to the innominate artery and tied within the origin of the vertebral, for no branch was seen between the bifurcation of the innominate and the ligature. On tightening the ligature the radial pulse was completely stopped and the aneurysm collapsed, and all pulsation ceased. The time occupied in the operation from first to last including suturing was eleven and a half minutes.

Within ten hours a faint pulsation could be felt in the radial, and on the day following the operation the pulsation was distinct.

On April 23 all stitches were removed and the wound healed, but pulsation in the sac had returned, although the aneurysm was smaller than before operation.

On April 28 the radial pulse was almost as large as before operation.

During the months of May, June, and July of 1899 the aneurysm continued to pulsate, the arm began to waste, and there was much pain owing to pressure on the brachial plexus.

Second operation—ligature of first part of the axillary artery.—On August 4 the first part of the axillary artery was tied in the interval between the pectoral and deltoid muscles. Some difficulty was experienced in finding the vessel, but it was ultimately secured with kangaroo tendon in about half an hour.

The radial pulse was arrested, and pulsation in the sac ceased.

On August 1—7 faint pulsation was detected in the radial artery.

On August 8 the sac of the aneurysm was much smaller, the radial artery was just perceptible, and the shooting pain down the arm persisted.

Healing of the wound was delayed on account of the difficulty in getting the skin to fall into the hollow, left after the dissection, beneath the clavicle. Aneurysm shows a return of pulsation.

The patient was now lost sight of for some months, as he lived at a distance, but on the advice of Dr. Harris he again returned to the Cumberland Infirmary on January 17, 1900. The chief complaint was loss of power in the right hand and arm, and pain shooting down the arm. The loss of power of the hand was marked, as the hand could not grasp anything, or the fingers close upon anything resting in the palm. Marked muscular wasting in the upper and forearm was present.

At the root of the neck, in the supra-clavicular space, there was a pulsating swelling forming a distinct protrusion some four inches in length and three inches in depth. The pulsation was expansive.

By deep pressure at the upper and inner part of the aneurysm pulsation in the sac could be controlled and almost obliterated. It was clear that the aneurysm was now pressing back on the brachial plexus, and forward against the collar-bone.

There was no alteration in the colour of the skin over the aneurysm, or sign of adhesion, and the collar-bone could be slipped over the aneurysm when the arm was raised.

There was no evidence of extension of the aneurysm into the axilla.

Third operation—excision of portion of clavicle and removal of sac.—On January 21, 1900, the clavicle was exposed and bared for some four or five inches, and two and a quarter inches of the middle and outer thirds sawn away.

The upper and lower surfaces of the bone were freed by a periosteal elevator, and the posterior surface of the bone required careful separation from the sac of the aneurysm. The portion of bone removed was sawn after first passing a copper retractor beneath.

The aneurysm was now in full view, and was seen to be fed by a large vessel at the upper and inner side of the sac.

This vessel emerged from behind the anterior scalene muscle, and passed almost perpendicularly downwards and forwards to the sac.

When this vessel was tied and divided the aneurysm shrank to a small size (from a hen's egg, or rather larger, to the size of a large walnut).

The aneurysm was then dissected away from the proximal towards the distal end, and the exit vessel finally tied with kangaroo tendon.

The operation was not a difficult one, neither was there

much blood lost, as all vessels cut were secured with a ligature or pressure forceps at once.

The pulse at the wrist was obliterated, at the wrist for the third time.

The cords of the brachial plexus gave no trouble, and the subclavian vein was not seen. The portion of clavicle cut out was not replaced. It should be added that in addition to the skin incision along the collar-bone it was found needful to make a second incision up the neck at right angles, and to turn the flaps back in order to get free access to the aneurysm. Duration of operation forty-five minutes.

At the first dressing on January 25 an extremely faint pulsation was felt in the radial artery, and the numbness in the hand was not only gone, but the fingers could be bent, and the patient grip fairly well.

On the 27th a sphygmographic tracing of the radial artery showed a slight undulating line, proving the re-establishment of collateral circulation in the limb. The return of power in the fingers and hand was now marked.

On January 29 the pulse was sufficiently good to admit of counting.

On February 5 it was noted that the power had quite returned to the hand.

The wound healed without trouble, and there was hardly any elevation of temperature.

Present condition as to arm.—All movements of the arm can be performed except elevation of the upper arm above the level of the shoulder.

Condition of clavicle.—The divided ends of the bone have approximated each other, and are only about an inch apart, but they are not on the same level; the outer end has been drawn upwards, but not more than an inch. The shoulder has fallen forwards somewhat, but this is not very noticeable.

General remarks.—The urgent need for doing something for the patient was referrible to the state of the arm, which had become useless as well as painful.

The ligature of the first part of the subclavian was only of temporary service, whilst the ligature of the artery below the collar-bone was of no service whatever.

The third operation, which was undertaken when no other course was open, was adopted by reason of the success attending this mode of procedure in the hands of Mr. H. W. Allingham, who, so far as I know, first planned and carried it out (*Lancet*, p. 1487, June 3, 1899).

Mr. Allingham tied the first part of the subclavian without success, and then cured his patient by excising the aneurysm, but he attached importance to the preliminary operation as tending to establish collateral circulation.

The aneurysm.—On slitting up the sac no vestige of clot was seen, and very little beyond greyish patches of thickening in the wall of the sac due to atheroma.

It must be recollected that about nine months' interval separated the ligation of the subclavian and excision of the sac, so that it is not surprising that no trace of the subclavian artery was found on the outside of the aneurysm.

With regard to the feeding vessel, which was about the size of a quill pen, and came downwards from behind the anterior scalene muscle, I cannot venture to say what artery it was.

Examination of the portion of collar-bone cut out shows that the posterior surface was worn but not eroded, as the bone is distinctly thinner at the place where the sac lay beneath, and the ultimate result would have been erosion and fracture of the superjacent bone unless other complications had ensued.

The operation chanced to be straightforward, and the only structures of importance divided were the external jugular vein and the omohyoid muscle, whilst the feeding vessel was found at the place where it was believed to enter the sac before operation. It is of course unlikely that another case would be so uncomplicated with difficulties as, for instance, in Mr. Allingham's case. The brachial plexus and subclavian vein created trouble, and this operation occupied two hours.

The notes of the first two operations were taken by Dr. Cairns, house surgeon at that time. The notes of the third operation are by Dr. Sage Sutherland, present house surgeon.

Professor Page, of Newcastle-on-Tyne, was present at the last operation, and gave me valued help.

DESCRIPTION OF PLATE XXIX.

To illustrate Sir Hugh Beevor's case of Adiposis Dolorosa.

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XXXVII.—*A case of Diffuse Lipoma (adiposis dolorosa).* By Sir HUGH BEEVOR. *Exhibited April* 27, 1900.

E. C., æt. 40, married, admitted to King's College Hospital, April, 1900. Of German parentage, father healthy, mother died of bronchitis, no history of similar ailment in the family.

Personal history.—Married fifteen years ago, one child survives the last born; he has undergone operation for large glands in the neck, axillæ, and groin. She had previously three miscarriages, a stillborn child and an infant which survived but ten days. Soon after marriage she became very stout, weighing eighteen stone; her stoutness then considerably abated, and seven years ago she began to notice this swelling of the limbs, which has continued to increase since then. Latterly pain of a sharp, shooting character and worse at night, has affected the limbs.

Present condition.—The legs are excessively stout down to the ankle; deep sulci from folding over of the fatty tissue appear at upper part of thighs, at knees and ankles. This excess of tissue extends over the buttock and to level of umbilicus in front, but not to feet nor elsewhere on the trunk. The upper limbs each show a large mass all along the posterior aspect of the upper arm.

The skin itself is not thickened; in some places it exhibits a granular kind of unevenness, in other places it seems thinned: there is no pitting. Hair and nails are normal.

Pain has not been a marked feature till now. Examination of the neck suggests a diminution in the size of the thyroid gland.

Her mental and bodily powers seem normal and unchanged. Nothing abnormal was discovered in examining circulatory, respiratory, alimentary, and renal systems.

After being six weeks under observation and taking thyroid extract she professed to feel improvement, but her weight and nearly all the measurements remained the same. The exception in the latter was the left calf, which increased some three inches in girth, appeared reddened, and was rather warmer than its fellow, at the same time giving rise to much burning sensation for a few days, relieved by belladonna

ointment. That this case agrees with those described already by Dercum and Collins may be seen by reference to the *British Medical Journal*, December 2, 1899 (case of Hale White).

That the type of disease is very distinct was evident from the fact that many physicians who saw this patient at the Clinical Society believed they had seen her before at other hospitals; they remarked upon her facies as much as upon the local symptoms to account for the mistaken identity.

Measurements.—Upper arm, four inches above olecranon: right, 15 inches; left, 15 inches. Forearm, four inches below olecranon: right, 11 inches; left, 11 inches. Thigh, four inches above spine of tibia: right, 25 inches; left, 25 inches. Leg, four inches below spine: right, 21 inches; left, 20 inches. Neck, 13 inches.

XXXVIII.—*A boy with (apparently) Tubercular Mediastinal Glands pressing on the Right Lung.* By NORMAN DALTON, M.D. *Exhibited April* 27, 1900.

A. H. L., æt. 12. When admitted to King's College Hospital the report was that there was a strong tubercular family history, but closer inquiry negatives this; the only tubercular case in the family being an uncle, who was also alcoholic. Except for diphtheria the boy was well until five months ago. Since then he has become thin, and has had a pain in the right axilla.

The physical signs are as follows:—There is no appreciable difference in the measurement nor in the expansion of the two sides of the chest. The heart is normal. There is dulness behind the upper part of the sternum, which extends towards the right for about one inch beyond the right border of that bone. Beyond this the resonance is impaired both above externally (as far as the axilla), and below internal to the nipple, but these parts are not absolutely dull. Over the dull area the heart's sounds are very loud, and breath-sounds of a bronchial character (though not pure bronchial breathing) can be heard. But external to the dull area the breath-sounds

are notably diminished compared with the left side, and the vocal fremitus is also greatly diminished. So that, to sum up, there is dulness over the sternum and the adjacent part of the right lung, with comparative dulness, deficient breath-sounds, and deficient vocal fremitus beyond the dull area. In addition the veins over the front of the chest, proceeding from the sternum, are dilated. When the head is retracted the veins in the neck become greatly distended, but no bruit can be heard over the base of the heart. Over different parts of the rest of both lungs an occasional crepitation can be heard, but nothing constant. The rest of the body presents nothing of importance. There is a hard, rather enlarged gland in the left axilla, and the abdomen is full, with slight enlargement of the superficial epigastric veins, but nothing can be felt there. The patient has been under observation for a fortnight, during which time there has been marked remittent fever; he has lost weight, and I think that the physical signs are more marked. For instance, at the first examination the dulness behind the sternum was not so definite.

As regards the diagnosis, since the dulness now extends behind the sternum the physical signs in the upper part of the right lung can scarcely be due to anything in the lung itself or to thickening of the pleura. It therefore seems clear that there must be something solid extending from the mediastinum and compressing the bronchus leading to the upper lobe of the right lung which would make the resonance, breath-sounds, and vocal fremitus deficient in that region. It is noteworthy, however, that the breathing at the back of the right apex is not less than at the corresponding position on the left side, so that apparently the air reaches that portion of the right lung more easily.

When it was thought that there was a strong tubercular history the conclusion was that the mediastinal mass must be composed of tubercular glands, particularly when the patient's appearance and his temperature were considered; but as there is no real tubercular history, and as, so far as can be ascertained, the mediastinal growth appears to be increasing in size rather rapidly, it is quite possible that the growth may be lymphadenomatous or sarcomatous, and growing from the mediastinum into the lung along the bronchus as such growths so often do.

N.B.—The differential count of the white blood-corpuscles, for which I am indebted to Dr. Whitfield, gave the following

results:—Polynuclear leucocytes, 66·5 per cent.; lymphocytes, 23·1 per cent.; large mononuclears, 5 per cent.; eosinophils, 3·2 per cent.; myelocytes, 1·1 per cent.; small myelocytes (probably dwarf forms), 1·1 per cent. The increased number of eosinophils and myelocytes favours the idea that the mediastinal growth is lymphadenomatous, but on several occasions the number of red corpuscles has been found to be normal. This complete absence of oligocythæmia is practically incompatible with lymphadenoma, as it remains most likely that the growth is composed of tubercular glands.

XXXIX.—*A case of Macular Leprosy improving under chaulmugra oil.* By T. D. Savill, M.D. *Exhibited April* 27, 1900.

THE patient was an intelligent lad æt. 14, who was born in Demerara. He had left that place and come to England at the age of eleven. The disease began when he was ten with erythematous and pigmented patches slightly raised, scattered in different parts of the body, for the most part symmetrically. He said that he had been unable to feel in most of these patches, and he had had other places where there was no pigment or erythema which were also without sensation. Small patches of pigment had been observed on the soft palate, but these had now quite disappeared. The patient's skin was inclined to be a little dark, and he presented the appearance of a half-caste, both his parents having been half-castes. Scattered over the arms and legs were a series of ovoid, slightly pigmented areæ. These areæ were now quite flat, and presented no alteration in texture at the present time. In many of them the pigment was rapidly disappearing. Both the ulnar nerves, but especially the left, were still somewhat thickened, though much less than formerly. The chief interest of the case lay in the remarkable improvement which had taken place under the administration of chaulmugra oil. He had taken this remedy for nearly a year in gradually increasing doses until now he was taking forty capsules, each containing four minims of the oil, per diem. For some months he had rubbed in an ointment made of the same oil, but this had been given up for the past five or six

months, and the improvement had been nevertheless continuous. The swelling and erythema had entirely disappeared. The pigment had now almost gone. No anæsthesia existed anywhere at the present time, and the ulnar nerves were decidedly less thickened than they used to be. He had never had any tubercular leprous lesions at any time.

XL.—*A case after Removal of the Entire Clavicle.* By Charters J. Symonds, M.S. *Exhibited April 27, 1900.*

Joseph G., æt. 47, a carpenter, was admitted into Guy's Hospital under my care on February 23, 1899, for a tumour of the left clavicle. He stated that four years ago he received a blow on this bone, and was unable to use his left arm for some months. Eighteen months ago he first noticed a hard lump about the size of a marble on the left clavicle, at the junction of the inner and middle thirds. Six months ago it began to grow rapidly.

On admission an oblong tumour occupied the site of the left clavicle. The outer third of the bone—the acromial end—could be distinguished. The growth was tense but elastic, was smooth in outline, without lobulations. There was no expansile pulsation. It involved the sterno-clavicular joint, and projected backward into the neck. The diagnosis made was that of central sarcoma. There was no pain, and no evidence of pressure on the vessels.

On February 27 the growth was removed by dividing the clavicle and dissecting towards the sternum. It proved to be encapsuled, and was easily and quickly removed. An extension behind the sternum gave rise to a little trouble, but it was not found necessary to excise any part of this bone. The microscope showed the growth to be composed of small round-cells chiefly, but all the other appearances were those of a central sarcoma. The union was delayed by suppuration of a blood-clot. The final condition of the man is one of full usefulness. He can follow all the branches of his trade as a carpenter, and possesses all the movements of the shoulder. The shoulder girdle does not appear in any way weakened, nor has the scapula fallen outwards.

XLI.—*A case of New Growth of Base of Skull, with unilateral Paralysis of Cranial Nerves—first, second, third, fourth, fifth, sixth, and twelfth.* By PERCY KIDD, M.D. *Exhibited April* 27, 1900.

MARY ANN G., æt. 55, married.

Nose asymmetrical, deviates to left (congenital).

Ptosis of left eyelid complete.

Left eye slightly more prominent than right, completely motionless. No strabismus. Pupil dilated and fixed. Sight of left eye entirely lost. Optic disc atrophied.

Right eye normal, movements unaffected. Pupil of medium size and reacts normally. Optic disc normal.

Sense of smell on left side very defective, not entirely lost.

Sensation of touch and pain very defective over left side of face, more especially in naso-labial and malar region.

There is also tactile anæsthesia of left conjunctiva, nasal and oral cavities, including the left side of the tongue; conjunctival reflex lost. Cornea clear.

Left masseter and temporal muscles paralysed and atrophied; contraction to faradism lost, very sluggish contraction to constant current. No facial paralysis.

Hearing defective on both sides, but the deafness is attributed by Mr. Hovell to chronic middle-ear disease, both tympanic membranes being opaque and retracted. Septum nasi bulging on both sides, obstructing nasal passages.

Taste appears to be slightly less acute on the left side, but the difference is doubtful. Soft palate moves freely. Faucial reflex lost on left side. No laryngeal paralysis. Tongue protruded slightly to left. No affection of limbs.

History.—Liable to headache for many years, worse the last twelve months.

Present illness began in October, 1899, with loss of flesh aad appetite, increased headache (occipital and frontal), and she accidentally discovered that she had lost the sight of the left eye.

In February, 1900, left eyelid began to droop, and she began to notice a "swollen" sensation on the left side of the face, forehead, and roof of the mouth, and her face began to sweat profusely from time to time.

Progress.—Headache has lessened considerably, and is less general. She now complains of a smarting, numb feeling in left naso-labial and malar regions.

XLII.—*A case of One-sided Paralysis of the Soft Palate, probably diphtheritic.* By StClair Thomson, M.D. *Exhibited April* 27, 1900.

PARALYSIS of the soft palate is always a subject of interest to the physician, and one-sided palatal palsy is still more so, for it is a rare if not a very rare condition. It usually comes within the province of the laryngologist, as it is almost invariably associated with paralysis of the vocal cord on the same side. The reason of this association is, of course, accounted for by the fact that both the levator palati muscle and some of the muscles which close the glottis receive their nerve supply from the same medullary nucleus, the nucleus ambiguus.

In the case shown, however, there was no question of central lesion, the affection being evidently a peripheral neuritis. The muscles of the larynx and tongue were quite unaffected. The history of the case was that six weeks previously the patient, a young man, had been laid up with double quinsy. He was attended by a medical man at his own home. Three weeks later he felt as if there was a lump in his throat, and fluids began to regurgitate through his nose. He was now shown to have paralysis and anæsthesia of the right side of his palate, which hung lower than the left. On stimulating the latter the whole soft palate was drawn up sharply to the left, producing a dimple on that side. The patient could blow out his cheeks, showing that there was sufficient power in the soft palate to prevent all the air from passing up and out through the nose. He still had some regurgitation of fluid if he swallowed hurriedly; and such words as "rub" and "egg" were pronounced as "rum" and "eng," showing that the post-nasal space was still incompletely shut off. The knee-jerks were normal, and the pupils equal and reacting. There was no albumen in the urine.

The chief interest of the case lay in its unilateral character. The palsy of the soft palate consequent on diphtheria was almost generally bilateral. It had been suggested by Dr. Beevor that the paralysis of the palate may have been double to begin with, and that one side was recovering quicker than

the other. If that were so, the rarity of the unilateral character now visible would have to be discounted.

The other point of interest was as to whether the case may have originally been one of simple tonsillitis without the Klebs-Loeffler bacillus. There were observers who had watched cases from their inception, and found that sore throats which had never shown the diphtheria bacillus were followed by palsy of the palate. In the present instance there appeared to be no means of verifying this. The history of the case was strongly suggestive of its being due to the diphtheria poison, although the persistence of the knee-jerks was uncommon, and the one-sidedness of the affection was very rare.

XLIII.—*A case of Aneurysm of the Subclavian Artery.* By J. Hutchinson, jun. *Exhibited April* 27, 1900.

A PATIENT æt. 50, with a fusiform aneurysm of the first and second parts of the left subclavian artery. There was evidence of pressure on the left recurrent laryngeal nerve, and a skiagraph showed marked dilatation of the heart. The heart-sounds were accentuated, and could be heard in the right axilla. There was only slight bruit to be heard over the aneurysm; the left radial pulse was smaller than the right one.

(Operation performed later proved that the aneurysm extended down into the thorax, and that it was impossible to place a ligature on the proximal side of the aneurysm. The first part of the left axillary artery was then tied, with some improvement in the size of the aneurysm and disappearance of the bruit. It was feared that this improvement would be of short duration, as a tendency to relapse showed itself. However, at the end of four months from the operation the aneurysm had shrunk so as to be hardly perceptible, and all his symptoms were greatly improved. There was still no pulse to be felt at the wrist.)

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